

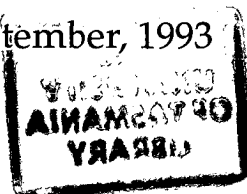
PSYCHOLOGICAL ADJUSTMENT AND  
SYSTEMIC LUPUS ERYTHEMATOSUS:  
ACOMPARATIVE STUDY

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## **SOURCES STATEMENT**

The present thesis describes original research undertaken in the Department of Psychology, University of Tasmania. To the best of my knowledge, any theories and techniques not my own have been acknowledged in the text. The remaining theoretical contributions in this thesis are my own original work and have not been submitted for any other degree.

Signed:

Helen Hornsby

## **ABSTRACT**

Systemic lupus erythematosus (SLE) is a chronic inflammatory disease of unknown etiology. It affects multiple organ systems and is characterised by periods of disease activity and remission. The unpredictable course, treatment and symptomatology of SLE can impact upon the social and personal resources of sufferers. Social resources are those variables influenced by external events, they include stress, uplifts, social support and social networks. Personal resources are mediated more by individuals' perceptions and include self-efficacy and coping (problem- and emotion-focused). Deteriorations in social and personal resources accompanying chronic illness suggest a Disease Exacerbation Model. This model proposes that the course of chronic illness is mediated by decrements in social and personal resources which, in turn, influence disease outcomes such as physical disability, psychosocial disruption and psychological distress.

The present investigation used a comparative design to test the Disease Exacerbation Model. The participants in the study were 34 individuals with a diagnosis of SLE, 37 multiple sclerosis (MS) sufferers and 38 people without a history of chronic illness. The control group was matched to the chronic illness groups for age, marital status, gender and socioeconomic status. Data were collected by using standardised psychological questionnaires. These included measures of stress, hassles, social network, social support, self-efficacy, coping, psychological distress, physical disability and psychosocial disruption.

Individuals with SLE and a chronic illness comparison group (MS) reported significantly fewer uplifts, less social support, more emotion-focused coping, as well as greater disability, distress and psychological disruption when compared with healthy people. There were, however, no significant decrements in network size or problem-focused coping and no significant increases in hassles. Except for the MS group reporting significantly more disability than SLE sufferers, no other differences were evident between the chronic illness groups. The correlations between social, personal and disease outcome measures suggest that group differences may involve somewhat different underlying processes. For example, social support mediated psychological distress for SLE sufferers, but not for the MS group.

To determine which social and personal resource variables are most salient to disease outcome, stepwise multiple regression analyses were performed. For SLE sufferers, increasing hassles and fewer uplifts were associated with elevated psychological distress. Although higher hassle levels and decreasing social support were both correlated with more psychosocial disruption, in the stepwise regression only hassles significantly predicted this disease outcome. Physical disability levels were not significantly related to any social or personal resource measures.

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## Chapter One

### Systemic Lupus Erythematosus: An Overview

### **1.1.0 INTRODUCTION**

This section reviews the medical aspects of systemic lupus erythematosus (SLE or lupus). It is included to familiarise the reader with the symptomatology, treatment and etiology of lupus. The chapter also provides the foundations for understanding research reviewed in later sections and discusses how the physical characteristics of SLE may precipitate psychological dysfunction.

#### **DESCRIPTION**

A concise and informative description of SLE is provided by Wallace and Dubois (1987);

“Systemic lupus erythematosus is a clinical syndrome of unknown cause or causes characterised by inflammation and multisystem involvement. It displays a widely variable presentation and course and is subject to multiple remissions and exacerbations in one or more systems. In approximately 30% of cases, the disease is induced by known drugs.”

(p.15)

#### **PATHOGENESIS**

To understand the pathogenesis of SLE it is necessary to briefly explain how the immune system functions. Whenever foreign organisms [antigens] such as viruses or bacteria invade, the immune system is mobilised. The first line of defence involves phagocytes engulfing

and digesting antigens (O'Donnell, Silove & Wakefield, 1988; Table 1). When the antigen is digested, phagocytes bind with antigen presenting cells. These latter cells then incorporate the nuclear material of the antigen into their membrane so it may be recognised by other immune defences.

Table 1: Cellular components of the immune system.  
(after O'Donnell et al., 1988)

-----	
1.	Phagocytes monocytes macrophages polymorphonucle neutrophils
2.	Antigen Presenting cells monocytes macrophages accessory cells
3.	T-lymphocytes regulatory cells helper cells suppressor cells effector cells delayed hypersensitivity cytotoxic T-lymphocytes
4.	Natural Killer cells
5.	B-lymphocytes plasma cells memory cells
-----	

When antigen presenting cells bind with T-lymphocyte helper cells interleukin-1 is secreted (Hardy, 1985). Interleukin-1 causes the proliferation of helper cells and these secrete interleukin-2 which has several functions. It stimulates T-suppressor cells that switch off the

immune response when the time is appropriate. Interleukin-2 also stimulates the reproduction of T-killer cells that will either bind with the antigen presenting cell or engulf the antigen directly. Finally, interleukin-2 stimulates B-lymphocytes to produce antibodies (immunoglobulins) that bind with the invading antigen so it is easily recognised by T-cells. B-lymphocytes are antigen specific and if the antigen invades again, they are mobilised immediately (Blau & Schultz, 1984).

Autoimmune diseases are characterised by immune attacks toward native body proteins. In lupus the immune attacks are directed toward deoxyribonucleic acid (DNA or the genetic material of cells). Since all cells contain DNA, every organ is a potential target for immune attacks. Although the etiology of immune attacks is not known, immune irregularities associated with SLE are well documented. Individuals with SLE have lower interleukin-2 levels, suppressor cells fail to switch-off the immune system and B-lymphocytes are constantly active whether or not there is infection (Blau & Schultz, 1984).

## **SYMPTOMATOLOGY**

Since all organs are at risk in SLE, there is no typical presentation of patients. Signs and symptoms of SLE are, however, characterised by inflammation and include arthritis, rashes, kidney involvement and fevers (Schur, 1983; Hardy, 1985).

The most common symptom of SLE is inflammation of the synovial membrane that surrounds the joints (Schur, 1983). This occurs in about 90 percent of sufferers and differs from rheumatoid arthritis in that joint deformity is rare. Often accompanying joint involvement is inflammation of the tendons and muscles which occurs in about 15 percent of lupus sufferers.

The skin is affected in over 70 percent of individuals with SLE (Hardy, 1985). Rashes may be present on the hands, feet or face and arise from inflammation of arterioles. A butterfly rash over the cheeks and nose bridge occurs in about 40 percent of patients. About 15 percent of individuals have discoid lesions. These are red, blotchy, scaly sores that may leave scarring upon healing.

Inflammation of the kidneys can result in protein and blood in the urine and is a significant cause of death in SLE sufferers (Schur, 1983). If the membrane that surrounds the lungs becomes inflamed, sharp stabbing pains on taking a deep breath may result. Pericarditis occurs when the pericardium membrane encasing the heart becomes inflamed. The symptoms of pericarditis mimic myocardial infarction and include shortness of breath, ankle swelling and breathing difficulties after exercise or when lying down.

Raynaud's phenomenon is similar to frostbite but occurs in the absence of cold weather (Blau & Schultz, 1984). It results from inflammation of arterioles that supply blood to the fingers and occurs

in about 15 percent of sufferers. Sjogren's syndrome occurs in 40 percent of patients and results from reduced gland secretions.

Other signs and symptoms of SLE include anaemia, seizures, psychoses, damage to the retina, temporary hair loss and mouth ulceration. Fevers, light sensitivity (photosensitivity), generalised aching and fatigue also are common. Gastrointestinal problems such as constipation, diarrhoea, nausea and vomiting are evident in 40 percent of sufferers (Blau & Schultz, 1984; Hardy, 1985).

Despite the diversity of SLE symptoms, rarely do individuals experience more than five or six of those described above. Furthermore, while organs such as the heart and kidneys may become involved, the majority of sufferers do not experience such complications. Regardless of the symptoms that arise, SLE can be debilitating as symptoms disrupt life-style causing sufferers considerable psychological distress.

## **DISEASE COURSE**

The symptomatology of SLE is characterised by periods of flare and remission (Blau & Schultz, 1984; Hardy, 1985). A disease flare occurs when symptoms increase in intensity and there is inflammation of the affected organs. Remission occurs when symptoms become more quiescent and inflammation is reduced. Since SLE is a chronic disorder, remission does not necessitate the absence of inflammation or symptomatology, rather symptom intensity is merely reduced (Hardy, 1985).

Since prognosis depends on numerous factors such as the severity of the disease, the organs effected, age and response to therapy, it is difficult to predict the course of SLE. Improved diagnostic strategies and pharmacological management are, however, contributing to an increasing survival rate, with the result that life expectancy has doubled in the last 20 years (Schur, 1983). In 1953, for example, the five year survival rate was less than 40 percent. In a 1987 study over 80 percent of individuals were alive nine years after diagnosis (Studenski, Allen, Caldwell, Rice & Polisson, 1987). Despite an increasing life expectancy and treatment advances, the major causes of mortality remain unchanged. These are progressive renal failure, central nervous system (CNS) symptoms and superimposed infections (Kinash, 1983; Studenski, et al., 1987; Wallace & Dubois, 1987).

Although SLE is sometimes life-threatening, the majority of individuals survive well into the sixth decade (Wallace & Dubois, 1987). Nonetheless, the prospect of kidney or CNS involvement or a poor prognosis remains a concern for many sufferers. Occasionally such concerns may disrupt social and occupational functioning and cause considerable psychological distress (Hardy, 1985).

## **DIAGNOSIS**

Systemic lupus erythematosus mimics the symptomatology of other diseases (Kinash, 1983). For example, it is common for individuals to present with evidence of arthritis, but there is no joint deformity



when x-rayed. Similarly, chest pains suggesting heart disease may be reported, but there are no electrocardiogram abnormalities. The pathogenesis and flaring and remitting course of SLE can make diagnosis difficult. Only when symptoms appear then disappear is the condition suspected and because symptoms can take years to appear, diagnosing the condition also may take years.

### ARACRITERIA

In 1971 the American Rheumatism Association (ARA) published preliminary diagnostic criteria to clarify the parameters of SLE (Wallace & Dubois, 1987). These criteria were revised in 1982 to achieve a diagnostic sensitivity and specificity rate of 96 percent (Schur, 1983). An individual has SLE if they meet four of the 11 criteria (Appendix one) and is given a probable diagnosis if they meet three. Symptoms need not be present simultaneously and a patient's medical history is considered when reaching a diagnosis. It takes an average of three years for a patient to meet four ARA criteria (Wallace & Dubois, 1987).

Despite the high reported sensitivity and specificity of the ARA criteria, the taxonomy has several limitations. It excludes important signs of SLE such as alopecia (hair loss), Raynaud's phenomena, persistent low grade fevers and fatigue. The criteria were derived from a small sample of SLE sufferers and from retrospective studies (Wallace & Dubois, 1987). The ARA scheme also may eliminate some individuals from an 'official' diagnosis, as it does not weight the importance of symptoms. For example, individuals could have

definitive symptoms such as immunological disorder, antinuclear antibodies and a discoid rash but would not be diagnosed with SLE as they met only three criteria.

Since the present study is not concerned with the medical diagnosis of SLE, the ARA scheme will be modified for recruiting volunteers. In this study, an individual has SLE if they meet (a) three ARA criteria and their treating physician has diagnosed SLE, or (b) have immunological disorder, antinuclear antibodies and one other ARA diagnostic symptom.

#### DIFFERENTIAL DIAGNOSIS

Discoid skin lesions in the absence of subcutaneous symptoms is diagnosed as discoid lupus erythematosus (DLE) rather than SLE. Discoid lupus is characterised by localised inflammation of the skin, often occurring in areas exposed to solar or ultraviolet irradiation. This condition is persistent but not life-endangering, although in some cases SLE develops (Hardy, 1985).

A lupus-like syndrome also may be induced by several classes of drug, including cardiovascular, antimicrobial, anticonvulsant and psychotropic medications (Schur, 1983). Drug-induced SLE differs from the idiopathic variety in several ways. It does not favour women more than men. Nephritis (inflammation of the kidneys) and central nervous system features are not ordinarily present. Antibodies to several classes of proteins are less common in drug-induced lupus. Finally, false-positive tests to syphilis (ARA

criteria 10) disappear when the offending drug is withdrawn (Krupp & Schroeder, 1987). In about 30 percent of individuals, however, the offending medications precipitate the idiopathic condition, suggesting some individuals have a pre-existing diathesis for SLE (Harmon & Portanova, 1982).

## **EPIDEMIOLOGY**

Several studies confirm the incidence of SLE is between 2.6–4.6 per 100,000 with a prevalence rate of 1 per 6780 (Meddings & Grennan, 1980; Wallace & Dubois, 1987). The ratio of women to men sufferers is reported as 9:1, although this varies with age. Below the age of 15 and above 60 this ratio is somewhat lower, with about twice as many females suffering from SLE as males. Explanations for the gender difference rates include loss of male siblings at birth, environmental and hormonal factors. These will be discussed further in the etiology section. Reported onset ages range from three months to 87 years, however most cases have their onset between 15 and 45 years. The average onset age is 28 years for females and 51 years for males.

Higher rates of SLE have been reported in black Americans, Hispanics, Chinese and Indian populations (Wallace & Dubois, 1987). Racial and geographic differences are however a likely consequence of sampling strategies and diagnostic practices. Studies comparing black with white Americans, for example, report threefold prevalence rates in the former group. Yet the prevalence rate for black Africans is comparable to that of white Americans. Another explanation for racial patterns stems from naturally differing levels of blood

constituents. Blacks, for example, have naturally higher levels of gamma globulins than whites (Wallace & Dubois, 1987). Differing SLE spectrum definitions also explain contrary prevalence rates. American studies that use ARA diagnostic criteria may exclude SLE sufferers, whereas Asian and Indian definitions are more inclusive (Wallace & Dubois, 1987).

## ETIOLOGY

While the pathogenesis is well documented, less is known about the causes of lupus. It is likely, however, that lupus arises from a combination of hormonal, genetic and viral causes. These are briefly considered below and the interested reader is referred to Wallace and Dubois (1987) for an extensive review.

## HORMONAL

Autoimmune conditions occur more often in women because they are more immunologically reactive than men (Talal, 1987). Immunologic reactivity is in turn regulated by sex [steroid] hormones. This observation was initially made in a species of hybrid mice (known as NZB/NZW) that spontaneously develops a syndrome analogous to human SLE. De-sexing the mice exacerbates SLE in pre-pubertal males but does not effect the disease course of females. Administering androgens to females, however, results in a normal life span and increases survival rates in mice already afflicted with SLE (Talal, 1987).

The regulation of immune processes by sex hormones is only possible if lymphocytes have androgen and oestrogen receptor sites. These exist on T-lymphocyte suppressor cells found in the thymus gland. Here both androgens and oestrogens decrease their reactivity. Receptor sites also exist on suppressor cells in the spleen and lymph organs. Here immunological reactivity is decreased by oestrogens and increased by androgens. Sex hormones also regulate interleukin-2 levels and thus killer and suppressor cell activities. The functions of interleukin-2 are diminished by oestrogens and increased by androgens (Bhalla, 1989).

## GENETIC

The inheritance of SLE is associated with the sixth chromosome of human cells which contains a region that controls immunological functioning. This area is generally known as the major histocompatibility complex (MHC) and in humans as the human leukocyte antigen (HLA) region. The HLA region has several subregions that are genetically determined. In the autoimmune disease rheumatoid arthritis (RA) for example, 75 percent of sufferers inherit a subregion known as HLA-DRw4 and occasionally the disease is associated with the DRw3 and DRw5 regions. Similar studies with SLE patients have been less successful in identifying associated HLA regions. DRw2 has been identified in 53.7 percent of SLE patients and 26.1 percent of controls and Drw3 is reported in 45.1 and 20.4 percent of SLE and control subjects respectively (Blau & Schultz, 1984).

## VIRAL

Viral ribonucleic acid (RNA) from myoviruses and paramyxoviruses have been consistently observed in SLE patients. High myoviruses and paramyxoviruses levels do not, however, imply a viral etiology as corticosteroid treatments suppress immune functioning making patients more susceptible to secondary viral infections (Blau & Schultz, 1984).

There remains, however, a general belief that viral agents are somehow active in SLE (Talal, 1987). This stems from several similarities between SLE and acquired immune deficiency syndrome (AIDS). These include the production of antinuclear antibodies, lower interleukin-2 levels, fewer natural killer cells, increase gamma globulin levels and depressed B-lymphocyte suppressor cell functioning.

## TREATMENT

Despite the progress medicine has made toward understanding the underlying symptomatology of SLE, practitioners remain disadvantaged when treating patients (Decker, 1983). The etiology of SLE is unknown and thus treatment can only focus on reducing inflammation and treating symptoms as they arise, altering medications as symptoms abate, intensify or change altogether (Blau & Schultz, 1984). The treatment of SLE is, therefore, highly individualistic.

## PHARMACOLOGICAL

Prior to the 1940's no drug or class of drug was helpful in treating the symptomatology of SLE. By the mid 1970's several classes of drug, including antimalarials, corticosteroids, immunosuppressants and non-corticosteroid anti-inflammatories had proved useful in managing disease symptomatology (Hughes, 1988).

### *Antimalarials*

Antimalarials came into use in SLE after their effectiveness against discoid lupus erythematosus had been observed. Although the mechanism of these drugs is unknown they inhibit antigen-antibody (ANA) formation, reduce light filtration by the skin (about 30 percent of disease sufferers exhibit photosensitivity) and inhibit viral replication (viruses have been implicated in the etiology of SLE). These drugs also reduce inflammation (Hughes, 1982). The most widely used antimalarials are hydroxychloroquine (Plaquenil) and mepacrine (Quinacrine).

There are, however, some side-effects that restrict the extensive use of antimalarials. For example, there is a high incidence of gastrointestinal disturbance, with nausea and vomiting, which is associated with all antimalarials. Other side-effects include premature greying of the hair, blotchy skin, convulsive seizures, myopathy (muscle weakness) and skin rashes. The most serious side-effects involve vision, usually blurring when the medication is started and a 'halo' effect around bright lights. Furthermore, deposits of these drugs collect in the cornea of the eye, causing tunnel vision

and possibly [irreversible] blindness (Hughes, 1982). Many of the harmful side-effects from antimalarials are avoidable, through careful monitoring and withdrawing the medication at the first signs of side-effects. Additionally, regular visits to an optometrist can reduce the incidence of visual impairments (Hughes, 1982).

### *Corticosteroids*

Corticosteroids are the most widely used drug in the treatment of SLE. While their mechanism is speculative, they are documented to decrease T cell numbers, interleukin-2 levels and natural killer cell activity (O'Leary, 1990). Like the antimalarials, however, there are several side-effects from prolonged use or high doses of corticosteroids. These include slowed hair growth, osteoporosis, cataracts, decreased concentration span, heightened senses, masking of infections, weight gain, elevated blood pressure, emotional problems, psychosis and diabetes mellitus to name a few (Blau & Schultz, 1984; Hughes, 1982; Sutton, Navarro & Stevens, 1984).

### *Immunosuppressants*

Immunosuppressants directly suppress immune regulation via decreasing lymphocyte production and interleukin levels. Their side-effects include nausea and vomiting. Since immunosuppressants suppress the immune functioning, they increase the risk of secondary infections such as pneumonia. Immunosuppressants also are known to interact with several common drugs, including alcohol, aspirin and some tranquillisers to cause other side-effects (Blau & Schultz, 1984).



### *Non-Corticosteroid Anti-Inflammatory Agents*

Mild cases of SLE may be treated with aspirin (acetylsalicylic acid). Aspirin contains an analgesic (pain reliever) and antipyretic (to reduce fever) and effectively reduces inflammation and fevers associated with SLE (Blau & Schultz, 1984). Side-effects from aspirin are often less severe than those of the other medications mentioned above and are often controllable. The risk of gastric side-effects, for example, may be overcome by administering the drug along with buffer solutions such as Alka-Seltzer (Hughes, 1982). There also are stronger forms of aspirin designed to manage disease flares. These are, however, used more cautiously as the risk of serious side-effects is greater. Possible side-effects include allergies, liver complications, intestinal bleeding and visual difficulties.

### *Summary*

With the exception of non-corticosteroid anti-inflammatory agents and low doses of corticosteroids, pharmacological treatments for SLE can be aggressive and cause substantial side-effects. Since the side-effects from medication can be more debilitating than the symptom they treat, it is not uncommon for individuals to experience considerable social and psychological problems.

### PSYCHOLOGICAL

In Australia, self-help groups and Lupus Societies provide the major sources of psychological management for individuals with SLE (Hardy, 1985). Individuals also may receive private counselling or psychological assessments as part of ongoing medical care.

Internationally there have been few published studies that evaluate the efficacy of psychological interventions with SLE sufferers. This is due to the limited understanding of how the disease effects the psychological adjustment of sufferers. Investigations of the social and psychological consequences of SLE would provide the foundations for intervention studies.

## **MEDICAL ASPECTS AND PSYCHOLOGICAL ADJUSTMENT**

The disease course of SLE may effect the social and psychological adjustment of sufferers in a variety of ways. In about 15 percent of sufferers, symptoms may directly cause psychopathology. For example, hypertension can induce psychotic states. The waxing and waning course of SLE may impede social and occupational functioning and prolonged disease flares may erode social support and self-efficacy beliefs. Symptoms that cause disfiguration such as facial rashes may precipitate depressive states. Side-effects from treatment such as weight gain also cause distress and corticosteroids can directly precipitate depression. Finally, because it takes an average of three years to diagnose SLE, individuals experience considerable stress in not knowing what they are suffering (Hardy, 1985).

### **1.1.1 SUMMARY**

This chapter provided a medical overview of SLE. The information presented provides the foundations for understanding literature reviewed in the next section. It also highlights the relationship between medical factors, social and psychological adjustment. In particular, symptomatology, diagnostic issues and treatment all can

contribute to social and psychological adjustment problems in sufferers.

## Chapter Two

### Psychological Adjustment and Systemic Lupus Erythematosus

## **2.0.0 OVERVIEW**

Chapter one established that immune system dysfunction is responsible for the symptomatology of SLE, which in turn can cause physical disability, psychosocial disruption and psychological distress in sufferers. The next section reviews research on the psychological and social factors related to SLE. In sections the review includes studies with other chronic illnesses, as there was insufficient research with SLE sufferers.

Consistent with other research, the chapter conceptualises factors affecting chronic illness as either social, personal or disease outcome measures (Hooker, Monahan, Shifren & Hutchinson, 1992; Revenson & Majerovitz, 1991). Social resources are those variables influenced by external events, they include stress, social support and social networks. Personal resources are mediated by individuals' perceptions and include self-efficacy and coping. Outcome measures are the consequences of chronic illness and include physical disability, psychosocial disruption and psychological distress (Husaini & von Frank 1985).

## **2.1.0 SOCIAL RESOURCES**

Stress, social support and social networks are important social resources influencing illness outcome. This section reviews how stress effects immune functioning, issues relevant to measurement and the role of stress in SLE. The section then focuses on social

support and social networks, relevant measurement issues and how they mediate disease outcome variables.

### **2.1.1 STRESS**

A concise and informative definition of stress is provided by Cox (1987).

“Stress, it is argued, can only be sensibly defined as a perceptual phenomenon arising from a comparison between the demand on a person and his ability to cope. An imbalance in this mechanism, when coping is important, gives rise to the experience of stress, and to stress response. The latter represents attempts at coping with the source of stress. Coping is both psychological (involving cognitive and behavioural strategies) and physiological. If normal coping is ineffective, stress is prolonged and abnormal responses may occur. The occurrence of these, and prolonged exposure to stress per se, may give rise to functional and structural damage. The progress of these events is subject to great individual variation.”

(p. 25)

### **STRESS AND IMMUNE FUNCTIONING**

Immune cells have receptors for stress related hormones such as, beta-endorphins, enkephalins, corticosteroids and catecholamines (Bhalla, 1989; O’Leary, 1990). These hormones affect immune functioning in different ways. For example, beta-endorphins enhance

overall immune functioning, while enkephalins stimulate T-lymphocyte and natural killer cell activity. Catecholamines (epinephrine and norepinephrine) cause the release of lymphocytes from storage and increase natural killer cell activity. Corticosteroids reduce T-lymphocyte numbers, impair interleukin-2 production and decrease natural killer cell activity.

Immune cells also manufacture hormones that influence CNS functioning. For example, interleukin-1 and interleukin-2 act on the hypothalamus and pituitary gland to raise adrenocorticotrophic hormone levels. Other CNS hormones synthesised by immune cells are substance P, beta-endorphins, enkephalins, corticosteroids and catecholamines (O'Leary, 1990).

Another line of inquiry investigates the relationship between exposure to acute or chronic stress and changes in immune parameters. Immune system responsiveness to acute stress was investigated by Zakowski and associates (Zakowski, McAllister, Deal & Baum, 1992). Following a stressful film, lower lymphocyte numbers were observed in 20 healthy men. This change was evident 15 minutes after stress exposure and lasted for about 90 minutes. A two week follow-up, however, reported that temporary lymphocyte alterations did not increase illness susceptibility.

Individuals exposed to chronic stressors, such as carers of Alzheimer's disease patients, also have impaired immune functioning. Kiecolt-Glaser and associates (Kiecolt-Glaser, Glaser, Shuttlesworth,

Dyer, Ogrocki & Speicher, 1987) found care givers had lower helper:suppressor cell ratios, T lymphocyte and T helper cell numbers than the comparison group. Although differences between natural killer cells and T suppressor cell numbers were not apparent, carers had higher titers to Epstein-Barr virus suggesting poor immune reactivity to antigens.

Since psychological variables can moderate stress responses, they also should mediate immune functioning and subsequent disease outcome. In one study, only individuals with low social support had impaired immune functioning (Baron, Cutrona, Hicklin, Russell & Lubaroff, 1990). This relationship was not mediated by either depression or stressful life events. However, a suitable comparison group was absent and social support levels were determined by a median split of the sample. Median splits have been demonstrated to confound social support with stress levels (see Thoits, 1985 for a review).

Social support levels also can mediate changes in immune functioning resulting from examination stress (Jemmott & Magloire, 1988). Salivary antibody (IgA) levels were assessed prior to, during and post examination time in 15 university students. Antibody levels were lowest during exam times and highest 14 days after exams finished. Students with higher social support had the lowest IgA levels and were in better health than the low support group. However changes in diet and sleep typically accompany academic stress, so these may have contributed to immune alterations (O'Leary, 1990).



Personal resources such as locus of control buffer the relationship between daily hassles and immune functioning (Kubitz, Peavy & Moore, 1986). Using a sample of individuals with either high or low reported hassle levels, no significant differences in IgA titers were evident. An internal locus of control, however, was associated with lower IgA levels. This suggests it is perceived control that effects immune functioning rather than stress. Nevertheless only one percent of IgA protein becomes anti-body reactive upon encountering an antigen making the index an unreliable assessment of immune functioning (O'Leary, 1990).

Another personal resource, self-efficacy also mediates the relationship between stress and immune functioning. Individuals provided with training to enhance self-efficacy reported less stress and showed increases in immune efficiency as measured by B-lymphocyte, T-cells and interferon levels (Wiedenfeld, O'Leary, Bandura, Brown, Levine & Raska, 1990). Although the study controlled for confounders of immune functioning, such as diet, menstrual cycle and circadian rhythms, its limitation was the exclusion of a suitable comparison group (O'Leary, 1990).

The cited studies suggest the immune system is highly reactive to both acute and chronic stress. They also suggest social resources (e.g., high social support) and personal resources (e.g., self-efficacy and locus of control) mediate the stress response and hence immune functioning. Nevertheless, it remains to be demonstrated how short-term immune alterations influence the disease process.

## MEASUREMENT

The previous section discussed the relationship between stress and immune functioning without regard to the controversy regarding stress measurement. The next section distinguishes between the different approaches and outlines the rationale for the measurement strategy adopted in the present study.

There are three approaches to measuring stress. The first conceptualises stress in terms of [physiological] responses such as heart rate and blood pressure. Measuring the physiological concomitants of stress arose from Selye's notion of a General Adaptation Syndrome (GAS; Cox, 1987). The GAS has three stages. During the first stage the body demonstrates changes characteristic of stress such as an accelerated heart rate and increased blood pressure. This phase is accompanied by decreased resistance to disease. In the second stage the body adapts to prolonged stress and resistance to disease increases. The final stage is characterised by exhaustion. In this stage the body's resistance to disease decreases and if stress is prolonged the organism becomes ill and may die.

Physiological response measures are not widely used in studies of chronic illness as they confound disease symptomatology with signs of stress. They also do not correlate with the three GAS stages, suggesting other factors mediate stress responses (Cox, 1987).

The second approach measures stress as a stimulus. This model assumes that external events have the potential to cause strain or

stress within a person. Stimulus models typically use either life event or daily hassle inventories as stress measures. Life events are major social changes that demand adaptation from an individual. Examples include divorce, death of spouse and birth of a child. Hassles are ongoing problems that cause social disruption. They include noisy neighbours and work stress (Chamberlain & Zika, 1990). The life events and daily hassle stress measures are the most popular approaches in chronic illness research because they quantify stress and are easily administered.

The final approach uses an interactional paradigm, assessing stress as a stimulus and a response (Cox, 1987). Research adopting this approach also measures variables that moderate the stress approach such as self-efficacy and coping skills. The distinctions between the different measurement approaches are however becoming less clear as researchers routinely measure a range of behavioural and cognitive factors that effect stress reactions. Whilst the stimulus approach is adopted in the present study, it is considered in the wider context of other cognitive and behavioural factors.

## **LIFE EVENTS AND DAILY HASSLES**

Since the stimulus approach is less likely to confound disease symptomatology with stress, it is used widely in chronic illness studies. Two extensively used measures are life event and daily hassle inventories. This section provides a description of each assessment approach and a discussion of their relative merits.

## LIFE EVENTS

These are assessed by assigning standardised weights for the amount of readjustment a given event requires. The weights are summed to yield an index of life change or stress. Higher life change scores are associated with an increase in psychological distress, psychosocial dysfunction and physical symptoms (see Felner, Farber & Primavera, 1983 for a review). Correlations between life change and disease are however small, ranging from .10 to .20 (Felner, et al., 1983). This suggests that other factors such as personal resources mitigate the relationship between stress and disease (Felner et al., 1983).

## DAILY HASSLES

A similar methodology assesses daily hassles as antecedent to disease outcomes. This approach has, however, notable differences to the life event method. It does not assume standardised stress scores, rather respondents estimate the amount of stress and pleasure (uplift) an event provides (Chamberlain & Zika, 1990).

Studies measuring both life events and daily hassles suggest the latter approach is a better predictor of disease outcome (Weinberger, Hiner & Tierney, 1987; Chamberlain & Zika, 1990). Daily hassles and uplifts account for more variance in concurrent and subsequent disease outcomes than do stressful life events. Daily hassle and uplift inventories also have stronger test-retest reliabilities for the reported number and rated severity of events, when compared with life event inventories (Chamberlaine & Zika, 1990). For the aforementioned

reasons the daily hassle and uplifts approach was adopted in the present study.

### **LIFE EVENTS, HASSLES AND SLE RESEARCH**

Only a few studies document the impact of stress on SLE and they do not typically measure variables mediating the stress/disease outcome relationship. The earliest was concerned with the ways stress contributed to the onset of SLE. Otto and Mackay (1967) defined stress as "the conscious experience of tension— that is, depression, frustration, anger or anxiety, or undue physical strain" (p.489). Twenty SLE volunteers and a comparison group of women who had an accidental haemorrhage during pregnancy were matched for age, sex and socioeconomic status. Stressful life events were assessed via a structured interview. Significantly more SLE (100%) than control (60%) subjects reported that stress preceded the onset of their condition. A further 65 percent of SLE patients reported that life events also had preceded a disease exacerbation. The most frequently reported life events involved interpersonal relationships for both SLE and control subjects.

The Otto and Mackay study has several methodological problems that detract from the value of its findings. The rater was not 'blind' to the groups' diagnoses and individuals recalled stressful life events that occurred as long as 15 years prior to disease onset. The average time since diagnosis was 6.5 years for SLE sufferers and 3.5 years for controls making the memory of the events surrounding illness onset more

reliable for the latter group. Finally, the researchers confound their stress and disease outcome measures.

More recent research has focused on the notion that chronic illness leads to elevated stress levels and this exacerbates disease flares. In one study, 80 percent of SLE volunteers reported they felt stress preceded disease flares and aggravated their illness (Laing, Rogers, Larson, Eaton, Murawski, Taylor, Swafford & Schur, 1984). This relationship was further investigated in a prospective study using life event methodology (Rimon & Kronqvist, 1988). Over a 3.5 year period, 50 percent of SLE sufferers reported one or more stressful life events preceded a disease flare. The most frequently reported events included loss of a spouse, serious illness of a close family member, marital crisis and financial difficulties. Although this study used a prospective design, a comparison group was not included. Thus, it is unclear whether life events occur more often in SLE patients than in the general population. The study also did not include personal resource measures such as self-efficacy and coping, to determine whether these mitigate stress levels and hence disease outcomes.

Using a daily hassles measure, Wekking and associates (Wekking, Vingerhoets, van Dam, Nossent & Swaak, 1991) investigated whether hassle levels were higher in SLE than RA sufferers. Whilst no differences in stress levels were apparent, stress was related to physical and psychosocial status for SLE but not RA sufferers. This study also has significant methodological problems. For example, the sample size was small and the assumptions of MANOVA were not met. The

effects of uplifts on well-being were not considered. A fatigue or boredom effect also was present, as subjects failed to complete the measures across consecutive assessment occasions. Furthermore, inclusion of a healthy control group would have allowed conclusions regarding whether a diagnosis SLE or RA in itself, leads to elevated hassle levels.

Given the methodological problems of the studies cited above, it remains to be established whether individuals with SLE experience greater stress levels than other chronically ill people or the general population. How social and personal resource variables mitigate the stress disease relationship also has not been reported. The present study adopts the daily hassle and uplift approach, to investigate group differences in stress and whether these measures predict disease outcomes.

## **SUMMARY**

Immunological studies demonstrate hormonal links between the immune and central nervous systems and psychological studies suggest that immune changes coincide with both acute and chronic stressors. Immune changes are however highly individualistic and may either increase or decrease upon exposure to stress.

Measuring stress as a stimulus, the reviewed studies suggest life events and daily hassles precede the onset of SLE and subsequent disease flares. These findings are however unimpressive when their methodological limitations are considered—most are retrospective,

uncontrolled or include only one comparison group. A two comparison group design is necessary for determining whether high stress levels are specific to SLE. There also is a need to investigate if stress predicts disease outcome when other social and personal resources are incorporated into the model.

### **2.1.2 SOCIAL NETWORKS AND SOCIAL SUPPORT**

Research has differentiated social networks from the functions of social support, though the terms are often used interchangeably. Social networks (or social embeddedness) can be divided into structural and interactional parameters (Cohen & Wills, 1985). Structural parameters include the number of network members and embeddedness in social organisations. Interactional dimensions include network composition (e.g., the relative number of friends, co-workers or relatives) and contacts between network members.

Social (or functional) support refers to a more specific idea than network. Its assessment involves identifying those aspects of social relationships that promote psychological and physical well-being. These include belonging, instrumental, self-esteem and informational aid (Cohen & Wills, 1985). Belongingness (also known as diffuse support and social companionship) is spending time with others in recreational or leisure activities. This may reduce distress by, for example, distracting individuals from worrying events or enhancing feelings of affiliation. Instrumental (or material or tangible) aid involves the provision of actions or materials, such as assistance with



work or providing money to pay bills. Receiving instrumental support may reduce distress by allowing the individual more time for other activities. Self-esteem (or emotional or expressive) support refers to communications or demonstrations that a person is valued. This promotes feelings of self-esteem and reduces vulnerability to stress. Informational (or appraisal) assistance includes the provision of advice and feed-back, that may aid in coping. It is likely that information support reduces stress by helping individuals to understand or define their problems.<sup>1</sup>

Since the distinction between social support and network has important assessment implications and explains contrary research findings, it is maintained throughout this text. The term 'social relationships' will describe the characteristics of social networks and support.

## MODELS

Social relationships may influence health in a variety of ways. Social resources may be mobilised only when an individual is, for example, ill or under stress. This is the buffering model of support and is shown statistically whenever an interaction between illness [or stress] and support is found (Cohen & Wills, 1985; Thoits, 1982). The peripheral model of support is a variant of the buffering hypothesis (Henderson, 1984; Cohen & Wills, 1985). It asserts that support influences health by helping with the recovery from an event after it

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<sup>1</sup> Although topologies distinguish support functions, research suggests they correlate (Cohen, Mermelstein, Kamarck & Hoberman, 1985).

has occurred. Evidence for the peripheral model also comes from a statistical interaction between support and stress.

Social relationships also may have a beneficial effect irrespective of whether an individual is ill or experiencing a stressful event. Evidence for this model comes from a statistical main-effect for support without the presence of an interaction effect (Cohen & Wills, 1985; Thoits, 1985). There is research to support both the main and buffering models and they are not mutually exclusive (Cohen & Wills, 1985).

#### **THE LINK BETWEEN SOCIAL RELATIONSHIPS AND DISEASE OUTCOMES**

Whether social relationships have a main- or buffering-effect, they protect individuals from stress and reduce the risk of physical and psychological ill health (Wallston, Alagna, DeVillis & DeVillis, 1983; Cohen & Wills, 1985). How social relationships influence health requires clarification. Proposed mechanisms include promoting healthy life styles, coping assistance and influencing physiological processes and therefore possibly disease outcomes.

#### **SOCIAL NETWORKS**

Social networks promote generalised feelings of psychological well-being that protect individuals from ill health. They provide members with a sense of predictability and stability, norms for behaviour, encourage positive affect and enhance feelings of self-worth and belonging (Cohen & Wills, 1985). Establishing and

maintaining social network ties is mediated by personal resources such as self-efficacy and the personality dispositions of network members (Monroe & Steiner, 1986).

## SOCIAL SUPPORT

Social support may influence disease outcomes by promoting 'coping assistance' (Thoits, 1986). Support functions help distressed individuals to cope by reinforcing their efforts to change the meaning, feelings, or management of stressful circumstances. Tentative evidence for this hypothesis comes from the noted similarities between topologies of coping and support. For example, problem-focused coping and instrumental support, both consist of attempts to remove or alter threatening environmental circumstances. Similarly, emotion-focused coping and emotional support, attempt to alter negative feelings that accompany distress.

Social support also may influence physiological reactions to stress. For example, one study measured support as a coping strategy and found it predicted 33 percent of natural killer cell activity (Levy, Herberman, Whiteside, Sanzo, Lee & Kirkwood, 1990). A study with Japanese living in Hawaii found low social support levels predicted high blood pressure independently of other risk factors such as smoking and alcohol consumption (Joseph, 1981 in Berkman, 1984). Social support also may moderate health related behaviours such as seeking medical advice, smoking, alcohol consumption and blood pressure (Levy et al., 1990). Finally, social support promotes adherence to complicated

medical regimes and life style changes (Gottlieb & Green 1984; Zimmerman & Connor, 1989).

## MEASUREMENT ISSUES

Social network measures can be divided into specific and global indices. Specific assessments ask about a single parameter of network structure, such as the number of significant others who potentially provide social support. Global measures simultaneously index connections with, for example, friends, neighbours and community organisations (Cohen & Wills, 1985). Network size is a better predictor of the main-effect model than are global measures (Cohen & Wills, 1985; Heitzmann & Kaplan, 1988).

Social support measures also can be distinguished along the global/specific dimension. Global measures ask about several dimensions of social support such as emotional, informational and instrumental aid. Specific measures ask about one functional aspect of social support, such as the context in which emotional support was received.<sup>2</sup>

Global social support measures usually yield support for the buffering model (Cohen & Wills, 1985). Specific measures only show a buffering effect if they coincide with support requirements. For example, instrumental support relieves financial stress.

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<sup>2</sup> This is also known as enacted support (Barrea, 1986)

Based on empirical findings (see Heitzmann & Kaplan, 1988 for a review), the present study uses a specific social network measure to investigate whether individuals with SLE have adequate access to potential sources of social support. A global measure of social support is also included to determine whether SLE sufferers have levels of functional support comparable to healthy and other chronically ill people. The main- and buffering- effect models are not investigated, as longitudinal data that control for pre-existing social support and stress levels are required (Thoits, 1985; Cohen & Wills, 1985).

#### **SOCIAL RELATIONSHIPS AND AUTOIMMUNE DISEASE RESEARCH**

Since the role of social relationships in the outcome of SLE has not been reported, the following discussion reviews recent research with other autoimmune diseases. Although depression is the outcome measured in most studies, several also consider disability and pain measures.

A study of multiple sclerosis (MS) sufferers found individuals with a progressive condition had larger social networks than persons with a relapsing-remitting disease course (Wineman, 1990). This finding suggests network size grows as disability levels increase and individuals rely on significant others for self-care. Whether social network members were potential sources of social support also was investigated. Perceived unsupportiveness from network members predicted depression in individuals with a progressive disease course. This finding was independent of demographic factors such as age, sex and socioeconomic status. Since the Wineman study was

cross-sectional, the possibility that depression limited access to social network members cannot be excluded.

The efficacy of the buffering and main effect models was examined prospectively by Brown and associates (Brown, Wallston & Nicassio, 1989). Rheumatoid arthritis sufferers completed questionnaires asking about emotional support, network size, depression, disability and pain on three assessment occasions spanning 18 months. Low levels of emotional support, but not the number of network members, were associated with elevated depression scores. This relationship was independent of pain severity, disability and demographic factors such as age and education level. Emotional support also interacted with pain severity. Individuals with high pain and low social support were more depressed than people with high social support and pain levels. This interaction was only present in a cross-sectional analysis of the data and not longitudinally. Furthermore, a path analysis of the data suggested that low social support levels resulted in depression which in turn decreased social support levels. This latter finding demonstrates how depression can confound social support levels.

Another study with 149 RA sufferers found that baseline social support levels predicted the severity of depression 15 months later (Fitzpatrick, Newman, Archer & Shipley, 1991). This finding was independent of initial depression and social support levels. This study used The Interview Schedule for Social Interaction which confounds network size with functional support, so it is not clear whether social relationship have a main or buffering effect.

A different research strategy looks at the positive and negative effects of social relationships (Revenson, Schiaffino, Majerovitz & Gibofsky, 1991). Revenson et al. found positive support exchanges predicted lower depression levels, while negative exchanges predicted high depression levels. The interaction between support and depression levels suggested that the positive aspects of support were not cancelled out by negative transactions. The highest depression levels were apparent in RA sufferers with high number of negative transactions and few positive social supports. A limitation of the Revenson et al. study is stress was not considered as covariate of social support satisfaction.

Social support and social networks can influence disease outcomes in individuals with MS and RA. This occurs through embeddedness in social networks and through the functional aspects of social support. It also appears that only positive social support exchanges are beneficial to psychological well-being. How personal resources, such as self-efficacy and coping strategies, interact with social support to predict disease outcomes has not been reported.

## **SUMMARY**

Studies suggest that high levels of social support protect chronically ill individuals from depression, disability and pain. Because these studies use correlational designs, it is not clear whether social support and social networks are similar in chronically ill individuals and healthy controls. This question is fundamental to SLE research, as there have been no published studies on social support and networks

of sufferers. How social networks and support interact with personal resources also is unclear. For example, individuals with high self-efficacy may better mobilise support networks and thus minimise the impact of stress on disease outcome.

## **2.2.0 PERSONAL RESOURCES**

Personal resources are those variables that are influenced by perceptions, they include self-efficacy and coping strategies. The next section reviews Bandura's notion of self-efficacy and the factors that promote efficacy beliefs. The limitations of self-efficacy theory are considered and a wider definition of the construct adopted. The operationalisation of self-efficacy is discussed, as well as how it differs from related personal resources. Finally, how self-efficacy interacts with autoimmune diseases such as SLE is considered. The second part reviews the Lazarus and Folkman (1984) coping model and those studies investigating coping in autoimmune diseases.

### **2.2.1 SELF-EFFICACY**

Bandura and associates (Bandura, O'Leary, Taylor, Gauthier & Gossard, 1987; Bandura, Cioffi, Taylor, Brouillard, 1988) assert both efficacy and outcome expectations mediate aspects of health behaviour. Self-efficacy expectations are individuals' beliefs about their capability of performing a specific behaviour in a given situation (Bandura, 1977). Outcome expectations are individuals' estimates that a given behaviour will lead to a specific outcome. The distinction between efficacy and outcome expectation can be clarified using an example.



Individuals may be sure that a particular slimming plan will reduce weight (outcome expectancy) but lack the confidence they can persevere with the diet (self-efficacy).

Integral to self-efficacy theory is the concept that expectations vary on magnitude, strength and generality (Bandura, 1977). Magnitude refers to the ordering of tasks by difficulty level. Given a hierarchy of tasks, persons with low magnitude expectations can perform only the simpler tasks. While individuals with high expectations feel they can complete most tasks. Strength is an individual's probability estimate of completing a task. Generality is the extent to which efficacy expectations generalise beyond a particular situation to other situations. For example, abstinence from alcohol achieved as an inpatient may not continue upon release from hospital.

Efficacy expectations develop from performance accomplishments, vicarious experiences, verbal persuasion and emotional arousal (Bandura, 1977). Most important for the development of efficacy expectations are performance accomplishment or learning from personal experience. Mastery of a difficult or feared task not only increases efficacy expectations, but also promotes skills for coping with problematic situations. Vicarious experiences are derived from the observations of other's successes and failures on a task and is analogous to behavioural modelling. To observe an individual successfully complete a task does not, however, ensure personal success on one's first or later attempts at the same task. For these reasons, vicarious experiences are less important to the development

of efficacy expectations than performance accomplishments. In verbal persuasion individuals are instructed that they can master a task. Since success or failure has not been personally experienced, verbal persuasion contributes only moderately to the formation of efficacy expectations. The final source of efficacy information comes from emotional arousal. Stressful situations can cause anxiety and depression that may impede an individual's task performance and lower efficacy expectations.

### THEORY REFINEMENTS

It seems appropriate to modify self-efficacy theory given recent empirical findings. Bandura (1978) maintains it is the expectation that behaviour cannot be *sustained* that mediates task performance. Overwhelming evidence suggests, however, that it is outcome expectations that mediate self-efficacy beliefs (e.g., Maddux, Sherer & Rogers, 1982; Marzillier & Eastman, 1984; Wang & Richarde, 1988). This can be illustrated by a hypothetical situation requiring a non-phobic individual to perform two identical tasks involving picking up a snake. In one task the snake is harmless and in the other poisonous. It is likely that self-efficacy beliefs will differ for the tasks (higher for the harmless than the poisonous snake) and these variations arise from the different outcome expectations.

The predictive validity of self-efficacy expectations also have been questioned. Investigations consistently demonstrate that past conduct predicts future behaviour more accurately than self-efficacy expectations (e.g., DiClemente Prochaska & Gibertini, 1985; Godding &

Glasgow, 1985), though this varies across different individuals and situations (Garcia, Schmitz & Doerfler, 1990). In fact, when past behaviour is statistically controlled the association between efficacy and future behaviour is not statistically significant (Garcia et al., 1990). Although Bandura (1978) acknowledges dispositional influences, situational factors are considered the primary mediators of self-efficacy. Empirical research suggests, however, that both dispositional and situational factors mediate self-efficacy expectations. When a situation is ambiguous, dispositional self-efficacy expectations are the best predictors of performance. If circumstances are clearly defined then task-specific ratings best predict behaviour (Wang & Richarde, 1988).

Psychometric difficulties also pervade the assessment of self-efficacy. The validity of self-efficacy ratings comes from their correlation with performance measures (Bandura, 1982). This logic has several problems. Ratings may be reactive due to the close temporal proximity of self-efficacy and performance assessments. The identical nature of efficacy and performance tasks also may lead to measurement redundancy. Finally, the high correlations between efficacy and performance assessments may be mediated by other factors such as self-esteem (Kazdin, 1978).

Self-efficacy theory requires some conceptual revisions, given its theoretical and methodological limitations. Self-efficacy is a cognitive construct influenced by outcome expectations and individuals' previous performance accomplishments. In new situations

dispositional self-efficacy expectations influence performance. The resulting outcome may modify dispositional efficacy expectations and allow individuals to predict their performance on subsequent [similar] tasks.

The aforementioned empirical modifications to self-efficacy theory have measurement implications. Since efficacy and outcome expectancies are highly correlated, this eliminates the need for separate assessments. In addition, dispositional measures predict a wider range of behavioural outcomes and allow comparisons between studies. The present study uses a dispositional measure for assessing whether SLE sufferers have lower self-efficacy and outcome expectations.

#### **RELATIONSHIP TO OTHER PERSONAL RESOURCES**

The concise operationalisation and measurement of self-efficacy depends on conceptual clarity. It is, therefore, necessary to distinguish self-efficacy from related personal resources. Health locus of control refers to individuals' attributions of whether their health is controlled by internal or external factors, whereas self-efficacy pertains to behavioural expectations (Wallston, Wallston, Smith & Dobbins, 1987). Internally oriented individuals have generalised expectations that their health is dependent on personal behaviour. Externally directed persons believe illnesses are unrelated to personal behaviour. Locus of control can interact with self-efficacy in different ways (Strecher, Devillis, Becker & Rosenstock, 1986). In a situation where control is possible, a person with high self-efficacy expectations copes with distress. If a highly efficacious person is denied control or coping

efforts continue in situations where control is not possible, then distress results. Individuals with low self-efficacy who are given control do not cope with stressful conditions. If low efficacious individuals are denied control over stressful events then distress may be minimised. These predictions are supported by empirical evidence (see Litt, 1988 for a review) and have implications for the interaction of self-efficacy with coping behaviour.

Attribution styles differ from self-efficacy beliefs, in that the former pertain to the causes of events and not behavioural expectations. If, for example, illness is attributed to external, specific and unstable circumstances coping will be satisfactory and distress minimal. If, however, illness is attributed to internal, global and stable factors coping will be less effective and depression may arise (Litt, 1988).

Self-esteem refers to individuals liking or respect for themselves, whereas self-efficacy pertains to performance capabilities (Litt, 1988). This distinction can be illustrated by an example. An individual can have high self-efficacy for completing a task but derive no increase in self-esteem from its successful accomplishment. Frequently, however, high self-efficacy and self-esteem occur together. That is, individuals develop high self-efficacy from activities that also promote self-esteem.

The coping process involves primary and secondary appraisals (Larazus & Folkman, 1984). Primary appraisals involve judgments of whether an event is stressful and secondary appraisals concern

possible courses of action. Secondary appraisals are complex and involve decisions about possible coping strategies, the likelihood of their success (outcome expectation) and whether the individual can apply these strategies (self-efficacy). Efficacy expectations are part of the secondary appraisal process (Strecher et al., 1986). Efficacy beliefs also are mediators of the duration and effort of coping behaviour (Bandura, 1977, 1978, 1982). Highly efficacious individuals use a wider variety of strategies to cope with the temptation to smoke than persons with low self-efficacy beliefs (Garcia et al., 1990). High self-efficacy expectations also are associated with adaptive problem-focused coping and low expectations with the use of less effective means of reducing distress, such as emotion-focused coping (DiClemente, et al., 1985).

The personal resources discussed above interact with self-efficacy beliefs to influence behaviour. For example, high self-esteem and self-efficacy beliefs usually co-occur. There are, however, clear distinctions between the reviewed personal resources and self-efficacy. These distinctions were emphasised to affirm that the present study is focusing on the specific concept of self-efficacy and not a wider definition adopted in some research.

### **SELF-EFFICACY AND AUTOIMMUNE DISEASE RESEARCH**

Studies with chronically ill individuals usually adopt wide definitions that only remotely resemble Bandura's notion of self-efficacy. The following review considers only studies that narrowly define

Bandura's notion of self-efficacy. These studies have used either RA or SLE sufferers.

A study of 101 individuals with rheumatoid arthritis found high self-efficacy scores were related to less functional disability cross-sectionally and prospectively after one year (Schiaffino, Revenson & Gibofsky, 1991). Strong self-efficacy beliefs also were related to the use of adaptive problem-focused coping and lower disability levels one year after the initial assessment. Self-efficacy was not related to depression on either assessment occasion. Pain levels did, however, interact with self-efficacy beliefs for predicting depression after one year. When pain was minimal, self-efficacy was not associated with depression; high pain levels accompanied by strong self-efficacy beliefs were related to elevated depression levels. Whether this latter finding was further mediated by problem-focused coping skills was not considered. While the Schiaffino et al. study demonstrates a complex interaction between self-efficacy and health, it is flawed by the assessment strategies used. The self-efficacy measure was specifically related to disability and thus not appropriate for predicting depression, coping or pain levels.

A complex relationship between self-efficacy and life satisfaction is also evident in the adjustment to rheumatoid arthritis (Smith, Dobbins & Wallston, 1991). Using path analysis the study found an internal locus of control was associated with high self-efficacy and greater life satisfaction, whereas the unavailability of instrumental social support was associated with low self-efficacy and high

depression levels. High life satisfaction levels were predicted by high self-efficacy beliefs, an internal locus of control and satisfactory instrumental social support.

An intervention study found that enhancing self-efficacy levels lead to changes in a range of cognitive and behavioural domains for rheumatoid arthritis sufferers (O'Leary, Shoor, Lorig & Holman, 1988). Enhancing self-efficacy beliefs decreased pain, disability, stress and depression levels and increased physical functioning. High self-efficacy levels also were associated with higher suppressor/cytotoxic T-cell numbers. Strengthening self-efficacy beliefs did not, however, enhance activity levels or improve immunological functioning. This latter finding may have been due to the confounding effects of medication on immune parameters. Another limitation was that conservative statistical approaches were not utilised in analyses- one-tailed significance tests were adopted and Bonferroni corrections for multiple comparisons were not considered. Thus it is difficult to conclude that increasing self-efficacy improves cognitive and behavioural functioning in individuals with rheumatoid arthritis.

A descriptive study with 201 SLE sufferers reported increases in self-efficacy over time (Braden, 1991). Since there were no experimental manipulations and no comparison groups were included, Braden's data only provides test-retest reliability of self-efficacy measure. Furthermore, the self-efficacy assessment used a visual analogue scale in which respondents were asked "How



satisfied are you with your ability to *control fatigue*" (Braden, 1991, p.162, italic added). No evidence of the construct validity of this measure was provided.

In summary, self-efficacy beliefs concern one's capability of performing specific behaviours. These beliefs arise from past behavioural accomplishments and the expected outcome of a situation. Empirical data suggest that self-efficacy mediates disease outcomes, such as depression, disability and pain, in individuals with autoimmune diseases. These studies have not, however, established whether self-efficacy levels differ between chronically ill and healthy people or whether self-efficacy is a significant predictor of disease outcome.

### **2.2.2 COPING**

The most widely accepted model of coping behaviour involves three stages; stress appraisal, perceptions of control and coping behaviour. While this model is well researched in psychology, it is less often adopted by medical researchers. This section reviews the Lazarus and Folkman (1984) coping model and reviews studies investigating coping in autoimmune diseases. The review does not include studies with SLE sufferers, as the author was unable to locate any published studies.

### **THEORY**

Lazarus and Folkman (1984) define coping as

"...the person's constantly changing cognitive and behavioural

efforts to manage specific external and/or internal demands that are appraised as taxing or exceeding the person's resources."

(p.141)

This definition asserts that coping efforts are dynamic, have cognitive and behavioural properties and are a response to a taxing situation.

The Lazarus and Folkman coping model proposes that distress results from how an individual appraises a stressful situation. The appraisal process has three stages. The first stage is primary appraisal and concerns judgments about whether an event is harmful. The next stage is termed secondary appraisal and is concerned with whether an individual perceives an event as controllable. In the third stage individuals employ emotional and behavioural strategies that alleviate stress.

Lazarus and Folkman also distinguish between problem and emotion-focused coping. Problem-focused coping consists of strategies aimed at problem solving or doing something to alter the source of stress. Emotion-focused coping is aimed at reducing or managing the emotional distress associated with a stressful event. While most stressors elicit both problem and emotional focused coping responses, problem-focused coping predominates when individuals feel they can actively reduce stress and emotional focused coping dominates when individuals believe that the stressor can only be endured. Other dimensions of coping such as maladaptive coping

and social support as coping assistance also have been identified (Thoits, 1986; Carver, Scheier & Weintraub, 1989).

The final assertion of the Lazarus and Folkman model is that coping is a dynamic process that reflects the contextual aspects of stress. While this notion has merit, coping also has dispositional qualities (Carver et al., 1989). That is, individuals have a preferred set of coping responses that they take into stressful situations. This study adopts a questionnaire measuring both the contextual and dispositional aspects of coping behaviour.

#### **COPING AND AUTOIMMUNE DISEASE RESEARCH**

Studies have focused on issues such as coping typologies, the use of downward comparisons as a coping strategy and the relationship of coping to depression, disability and pain. This section focuses on studies of individuals with rheumatoid arthritis, as there has been no research with SLE sufferers. Most of the cited research does not incorporate current theoretical notions of coping and is not theory driven.

A study with 158 outpatient RA sufferers used cluster analysis to group individuals according to the coping strategies they most typically used (Newman, Fitzpatrick, Lamb & Shipley, 1990). The mostly widely used strategy was passive coping. Passive copers did not adhere to any particular strategies but used a wide range of responses to a moderate extent. Another group used both emotion and problem-focused strategies to cope with their RA and associated pain. The third group

were active in coping with their condition. They rarely used social support as coping assistance. The final group consisted of RA sufferers who utilised social support or religion to cope with their condition. These empirically derived groups were not distinguishable on demographic factors, psychopathology, social support or disease activity. Notable differences in disability scores were, however, apparent between the groups, with active coping group reporting lowest levels. While the results of this study are encouraging, the measure of coping was devised on an ad hoc basis from existing scales and its reliability was not established.

Another study examined downward and upward comparisons as a coping mechanism in individuals with RA and mothers of acutely ill newborn babies (Affleck, Tennan, Pfeiffer, Fifield & Rowe, 1987). Downward comparisons involve viewing others as less fortunate than oneself and upward comparisons concern viewing others more favourably than oneself. The notion that downward comparisons are common in RA and mothers was supported. Only rarely were upward comparisons made. The majority of respondents, however, did not use either downward or upward comparisons as a coping strategy. The descriptive nature of this study and the fact that raters were not blind to the hypotheses make the results unreliable.

The Coping Strategies Questionnaire (CSQ) conceptualises coping as either cognitive or behavioural (Beckham, Keefe, Caldwell & Roodman, 1991). Using this questionnaire Beckham and associates found that RA sufferers using pain control and rational thinking as

coping strategies reported less depression, disability, hassles and pain than individuals not using such strategies. This relationship was independent of demographic factors including age and gender and medical variables such as disease severity. Since this study used a correlational design, the direction of the coping and adjustment relationship cannot be inferred. Furthermore, the cognitive coping dimension of the CSQ confounds personal resources, such as self-efficacy and self-esteem, with the process of coping.

The relationship between coping strategies, pain and depression was investigated by Brown and associates (Brown, Nicassio & Wallston, 1989). The subjects were 287 RA sufferers who had been diagnosed for seven years or less. Two dimensions of coping were examined, passive (or emotion-focused coping) and active coping (or problem-focused coping). Cross-sectionally and over a six month period, an interaction between passive coping, pain and depression was present. Individuals who were passive copers with high pain levels were more depressed than people not using such coping strategies. Individuals who were active copers reported low depression and pain levels when compared with passive copers. Since a correlational design was utilised, causality problems also exist with this study. While passive coping in the presence of pain may lead to high depression levels, depression may lead to poor coping and elevated pain levels. There also was a problem with the coping measure use in the Brown et al. study. The range of scores for passive coping was greater than for active coping, potentially biasing the assessment of the former coping dimension.

Furthermore, without the inclusion of a depressed pain comparison group, few conclusions about result specificity are possible.

A longitudinal study with 45 RA sufferers used the Ways of Coping Questionnaire (Lazarus & Folkman, 1984), to assess its relationship with self-efficacy, disability and affect (Revenson & Felton, 1989). More individuals relied on emotion-focused coping than on problem-focused strategies. While coping was not associated with either disability, self-efficacy or negative affect, it was associated with positive affect. Individuals using problem-focused strategies were more likely to report positive than negative affective states. Like the aforementioned studies, the correlational design lead to cause and effect problems. Also the effects of time-1 affect, disability and self-efficacy were not partialled out when predicting time-2 coping, thus confounding existing adjustment with subsequent coping strategies.

While this review is not comprehensive, it highlights the limitations of studies of coping in chronic illness sufferers. Most studies use questionnaires without established psychometric properties, instead choosing to derive coping measures ad hoc. Furthermore, few investigations assess whether there are characteristic differences in coping responses between different types of illnesses and whether ill individuals cope differently from healthy controls.

In sum, the Lazarus and Folkman coping model is widely cited in psychological research but rarely is incorporated in studies with

chronically ill people. The model proposes that coping occurs as a three stage process and distinguishes between emotional– (passive) and problem–focused (active) coping strategies. Although coping is hypothesised as situation specific, it also has dispositional elements. How individuals with SLE cope compared with other chronically ill and healthy people has not been reported nor has the interaction of coping with disease outcome measures.

### **2.3.0 DISEASE OUTCOME MEASURES**

These measure the consequences of chronic illness and include psychological distress (or psychopathology), physical disability, psychosocial disruption (e.g., communication limitations) and life style disruption (e.g., eating difficulties and recreational restrictions). However, only one SLE disease outcome measure, psychopathology, has received substantial research. The effects of SLE on the other outcome measures has not been reported.

#### **2.3.1 PSYCHOLOGICAL DISTRESS**

High psychopathology (or psychological distress) rates have been well documented in SLE sufferers and can be arbitrarily classified as either organic or adjustment syndromes. Organic or neurological syndromes arise from the pathogenesis of SLE and include seizures and cognitive decline. Adjustment reactions emanate from problems associated with living with SLE. For example, the unpredictable disease course may result in depression, fatigue may lower sufferers resistances to stress and weight gains associated with using corticosteroids may lower self-esteem.

## INCIDENCE AND SYNDROMES

A study by Rimon, Kronqvist and Helve (1988) found a psychopathology rate of 19 percent. The most common syndrome was depression in 13 percent of SLE patients. Depressive symptoms ranged from mild to severe cases, with neurotic depression most frequently diagnosed. Organic brain syndromes were present in 10 percent of individuals. The psychiatric status of 83 percent of the original sample was followed longitudinally for three years. Over this period, 36 percent of individuals with SLE experienced depressive illness and 16 percent organic brain syndromes. These syndromes persisted during the entire follow-up period. Although the Rimon and associates' study is prospective, psychiatrists were not 'blind' to patients' previous psychiatric histories and a comparison group was not included.

An investigation of psychopathology rates prior to and after the onset of SLE as well as cross-sectionally during an interview shows the latter approach yields the highest estimates (Lim, Ron, Ormerod, David, Miller, Logsdail, Walport & Harding, 1988). The sample consisted of 40 sufferers (36 outpatients and 4 inpatients) and a comparison group of 14 RA sufferers and 13 inflammatory bowel patients. Six SLE and no control patients reported signs of psychopathology before the onset of their condition. Using DSM-III criteria, three SLE patients experienced anxiety conditions, two major depressive disorder and one an organic brain syndrome. Twenty-five SLE patients and eight controls reported psychopathology after the onset of their condition. For individuals with SLE, major depression was present in 16 instances, anxiety in four cases and atypical psychoses in five individuals. At a cross-sectional



interview 40 percent of SLE and 30 percent of controls were identified as 'psychiatric cases'. From the interview, two SLE patients had psychotic symptoms, eight had major depression and six anxiety disorders. Concurrent measures indicated that psychopathology was associated with high stress levels, but not with objective indices of disease activity. Furthermore, psychopathology was not associated with the presence of neurological changes.

Psychiatric interviews and psychometric tests were used to quantify psychopathology rates in 30 outpatients and 49 inpatients with a diagnosis of SLE (Kremer, Rynes, Bartholomew, Rodichok, Pelton, Block, Tassinari & Silver, 1981). Forty-six percent of the sample were judged to have some current psychopathology, though the degree of disturbance was only mild. A sample of 37 individuals completed the MMPI, of this group 61 percent were classified as showing psychopathology. Scores were elevated on the hypochondriasis, depression and hysteria scales, a profile that is usually associated with neurotic concerns. These findings were unrelated to disease severity, neurological involvement or corticosteroid dosages. The efficacy of psychiatric interviews and psychological testing cannot, however, be evaluated as only a subsample completed the MMPI.

Elevated depression, hypochondriasis and hysteria scores on the MMPI also were found by Liang and associates (Liang et al., 1984). This profile pattern was unrelated to disease duration, stressful life event scores or whether individuals were diagnosed with SLE or RA. For individuals

with SLE, however, depression was correlated with high social disruption scores on the life events inventory.

Standardised psychological tests also were employed by Allen and Glicksman (1986) to assess psychopathology in SLE patients. This study found similar Profile of Mood States (POMS; McNair, Lorr & Doppleman, 1971) between individuals with SLE and the norms for healthy subjects. These groups in turn were significantly different from psychiatric patients who had elevated POMS profiles. One explanation for the contrary findings of the Allen and Glicksman study is they used a community sample of individuals with SLE while other studies use outpatient or hospitalised sufferers. A more likely explanation, however, concerns design flaws with the study. Only 22 percent of individuals approached volunteered for the study and no attempt was made to confirm these people met the ARA criteria for SLE. The POMS also is not considered a measure of psychopathology but rather an assessment of affect (McNair et al., 1971). There also were no appropriate control groups, rather SLE sufferers were compared with POMS norms.

In sum, the psychopathology rate for SLE sufferers varies between 19 and 71 percent. This large discrepancy arises from measurement strategies and the classification of psychological states.

## **ETIOLOGY**

The preceding review suggested that for about 10 percent of sufferers psychopathology has an organic etiology and in five percent of SLE

patients it arises from the side-effects of medication. Over 85 percent of psychopathology is reactive and arises from factors that are not well understood.

## ORGANIC

Psychopathology can result from disease complications such as hypertension and renal dysfunction. When this occurs, the administration of corticosteroids reduces disease severity and symptoms of psychopathology (Adelman, Saltiel & Klinenberg, 1986). Changes in cerebral blood flow occasionally cause psychotic symptoms in sufferers (Adelman, et al., 1986). Immune deposits in the choroid plexus of the brain also are implicated in the etiology of psychotic episodes. Similar deposits are reported in the brains of individuals with schizophrenia, leading to the notion that SLE may provide a model for schizophrenia. Autopsy studies report enlarged brain sulci in individuals with a history of psychiatric symptoms, but these observations are based on small samples and are not consistently replicated (Adelman, et al., 1986). Fatigue also is a common symptom of SLE and can lower depression and anxiety thresholds.

Corticosteroid medications cause signs and symptoms of psychosis in approximately five percent of individuals with SLE. Individuals with preexisting psychopathology or organic brain diseases are at greater risk for steroid induced psychosis than are people without such a history (Wallace & Dubois, 1987). The notion of a steroid induced psychosis is however controversial. Some evidence suggests that psychotic symptoms can improve following administration of corticosteroids.

Furthermore, withdrawal of steroid does not always lead to the symptoms of psychosis abating.

## ADJUSTMENT REACTIONS

The majority of psychological syndromes are reactive and probably result from changes in social functioning or the erosion of personal resources. Limited research has documented that elevated stress levels precede psychological distress and that self-efficacy training lowers distress. Research with other autoimmune diseases suggests that decreases in social support and emotion-focused coping strategies also elevate psychological distress levels. Clearly further research on variables moderating psychological distress is required.

### 2.3.2 FUNCTIONAL DISRUPTION MEASURES

Figure 1 provides a summary of the different approaches for assessing health outcomes. Levels 1–4 are objective measures and are used mainly in epidemiology studies to assess disease outcomes. Level one assessments quantify self-reported symptoms of disease such as headaches or somatic complaints. Level two measures the incidence or prevalence of disease as an outcome. The third level is an indirect disease outcome measure and is based on parameters such as prescriptions or surgical procedures. Level five, estimates health outcomes from mortality rates.

A different approach estimates functional disruption arising from illness (level four). In this approach performance is classified as (1) physical such as changes in ambulation, mobility and body movement

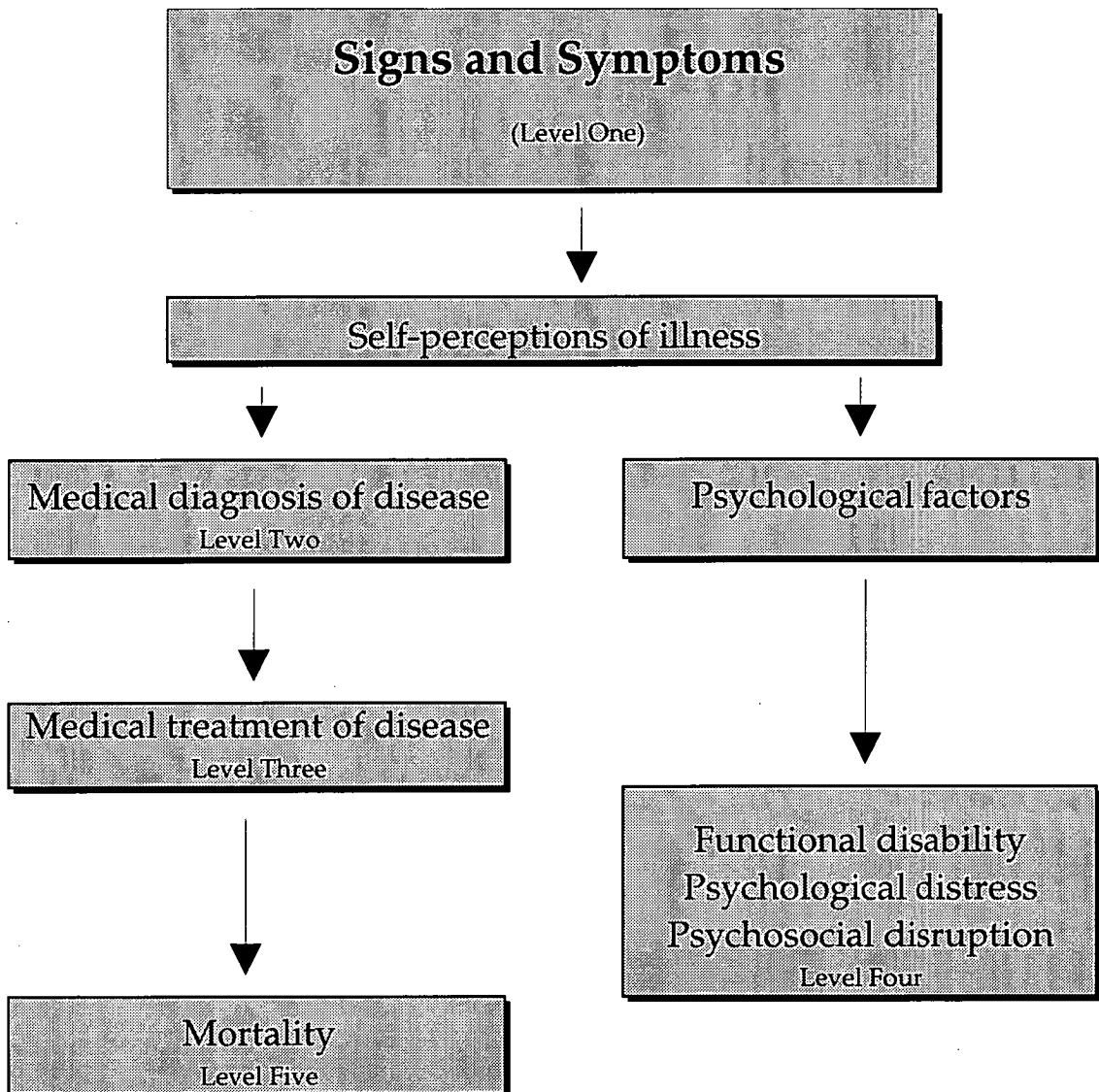


Figure 1: Medical approaches for assessing health outcomes  
(Modified from Bergner, 1988, page 82)

(2) psychosocial which includes disturbances in emotional behaviour, communication and alertness behaviour and (3) life style covering impairments in sleep, recreation, work and home management. The correlation magnitude between objective (levels 1–3) and functional approaches is typically moderate suggesting they measure distinctive aspects of disease outcome. In behavioural research, however, functional disruption measures have several advantages over the objective approach. A range of medical complaints can be compared irrespective of symptomatology or treatment. They also are sensitive to improvements in patient activity levels and are easily administered as self-report measures.

Only one study has measured the relationship between physical disability, psychosocial impairment and stress in SLE sufferers (Wekking et al., 1991). For individuals with SLE, but not the RA group, high stress levels were associated with elevated physical disability scores (i.e., mobility, physical ability, dexterity) and greater psychosocial disruption (i.e., anxiety, depression and social interaction) as assessed by The Arthritis Impact Measurement Scales (Meenan, Gertman & Mason, 1980). Disease outcome measures also were significantly correlated with objective indices such as auto anti-body levels and erythrocyte sedimentation rate (an index of inflammation and hence disease activity). Unfortunately, the study used a small sample size ( $n=13$ ) so the findings should be viewed with caution.

## **2.4.0 CHAPTER SUMMARY**

Consistent with earlier studies, disease outcomes are postulated to be

influenced by social and personal resources. Social resources are those variables influenced by external events and include stress, social networks and social support. Personal resources are mediated by perceptions rather than external events and include self-efficacy and coping strategies. The outcome of chronic illness includes indices such as psychological distress, physical disability or psychosocial disruption.

The social resources reviewed in this chapter were stress, social networks and support. Stress levels were similar in SLE and RA sufferers but higher than for 'healthy' controls (Wekking et al., 1991; Otto & Mackay, 1967; respectively). Furthermore, stress was related to disease outcomes for SLE sufferers, but not for people with RA (Wekking et al., 1991). Correlation research adds an additional dimension to the comparative studies. One study found that stress arising from a loss of social support exacerbated disease activity for SLE sufferers (Laing et al., 1984). The aforementioned conclusions are however based on a synopsis of findings from studies using different respondents and methodologies. A more advantageous approach would be to incorporate comparative and correlational designs to investigate stress levels and disease outcomes in the same group of subjects.

Comparative studies of social network size and support levels for SLE sufferers are lacking. One correlational study reported that the loss of social support in the presence of high stress exacerbated disease activity (Laing et al., 1984). But the study did not test the buffering model of social support. Correlational studies with other chronic conditions,

however, generally do not find a main-effect for social networks (Brown et al., 1989), but show a buffering effect for social support (Brown, et al., 1989; Fitzpatrick et al., 1991; Revenson et al., 1991). Since the affects of social relationships on disease outcome are not widely reported for SLE sufferers, it would be advantageous to adopt both comparative and correlational procedures to investigate these hypotheses.

The personal resources reviewed in this chapter included self-efficacy and coping. Comparative studies of self-efficacy have not been widely reported for SLE or other chronic illness sufferers. Several correlational studies have found that high self-efficacy perceptions are associated with a large social network, more social support and the use of problem-focused coping strategies (Wiedenfeld et al., 1990; Schiaffino, 1991; Smith et al., 1991). Again, comparative and correlational studies reporting the interaction of self-efficacy with other social and personal resources, as well as how it relates to disease outcomes are required.

Chronically ill people use more emotion-focused coping than problem-focused strategies. Whether they differ from 'healthy' controls has not however been reported. In correlational studies, illness sufferers using more problem-focused coping strategies report less disability and distress than those people who rely on emotion-focused strategies. Furthermore, problem-focused copers report fewer hassles and higher self-efficacy than people using



emotion-focused strategies (DiClemente, et al., 1985; Schiaffino, 1991; Smith et al., 1991).

The disease outcomes reviewed in this chapter were psychological distress, physical disability and psychosocial disruption. How social and personal resources influence disease outcomes for SLE is not well documented. For example, the incidence of psychopathology is widely reported and is believed to stem from adjustment problems associated with SLE. Nonetheless, how social and personal factors contribute to psychological distress in SLE sufferers is not well documented. Using physical disability as a disease outcome measure, one study found a correlation with elevated stress levels in SLE sufferers (Wekking et al., 1991). Thus there also is a need for comparative studies to establish where differences result on outcome measures and correlational research to determine which social and psychological resources may influence outcomes.

Throughout the chapter emphasis was placed on the need for comparative and correlation studies. The present study, therefore, includes another illness comparison group and healthy controls. Multiple sclerosis (MS) was chosen as the other illness comparison group for several reasons. It also is an autoimmune disease that occurs more often in females than males and has its onset during the child bearing years. The disease course of MS is characterised by flare and remission periods and exacerbations are usually managed with corticosteroids. Sufferers also are likely to experience their symptoms several years before a diagnosis is reached. The important difference

from SLE, is that antibody attacks are specific to the myelin sheath encasing central nervous system neurons. This difference in symptomatology, rather than the demographic characteristics of the diseases, will explain any observed disparities between the illness groups. The inclusion of a healthy control group allows conclusions about whether group differences are specific to chronic illness. The three group comparative design makes a useful contribution to research as it identifies and quantifies which social and personal resources are adversely affected by having SLE. The design also allows conclusions about whether changes are specific to SLE or occur in other chronically ill people.

The study design also allows for correlational and regression analyses to examine the relationship between variables cross-sectionally. While these analyses will generate hypotheses for further research, without longitudinal data firm conclusions about directionality and causality are not possible. For example, decrements in the availability of social and personal resources may result from, or contribute to, disease exacerbation.

To conclude, the literature suggests a Disease Exacerbation Model for the course of the disease and its impact on social and personal resources. This model predicts that the course of the chronic diseases will lead not only to a deterioration in the disease outcome measures, but also to a deterioration in various measures of social and personal resources. It is also to be expected that there will be some underlying casual relationships in which a diminution of certain social and

personal resources will impact upon the disease process further exacerbating the outcome as indicated by the distress, disability and disruption measures.

## **2.5.0 HYPOTHESES**

### **GROUP DIFFERENCES**

Following from the literature review, there is insufficient evidence to predict differences between the SLE and MS groups on any social, personal or disease outcome variable, except that the MS group may be expected to report greater physical disability. In accordance with the Disease Exacerbation Model, healthy controls may be expected to differ from chronic illness sufferers on all the social, personal and disease outcome measures. Expected differences between healthy controls and the chronic illness groups are as follows:

- a. The illness groups will report more hassles and fewer uplifts than healthy controls.
- b. The social network size of healthy controls will be larger when compared with SLE and MS sufferers.
- c. SLE and MS sufferers will report less social support when compared with healthy controls.
- d. Self-efficacy levels will be higher for healthy controls than the chronic illness groups.
- e. Controls will report more problem-focused coping than either SLE or MS sufferers.
- f. The chronic illness groups will report more emotion-focused coping than healthy controls.

- g. The chronic illness groups will report more psychological distress than healthy controls.
- h. The chronic illness groups will report more physical disability than healthy controls.
- i. The chronic illness groups will experience more psychosocial disruption than healthy controls.

To assist in interpreting group differences, correlational analyses also will be performed to identify relationships between personal, social and outcomes measures. Stepwise regression will be used to investigate the relative importance and strength of social and personal resources in predicting disease outcomes.

## Chapter Three

### Method

### **3.1.0 DESIGN**

The design of this study involved the comparison of three matched groups: SLE, MS and healthy controls. The groups were matched for age, gender, marital and socioeconomic status. The dependent variables used in the study were measures of social resources, personal resources and disease outcomes. Social resource measures included hassles, uplifts, social networks and social support. Personal resource measures were self-efficacy, problem- and emotion-focused coping. Disease outcome measures were physical disability, psychological distress and psychosocial disruption. A description of the various measures and the psychometric properties is provided in section 3.3.0.

The hypotheses were subsequently evaluated by analysis of variance to assess differences in group means on the various dependent variables. Correlation and regression analyses were also performed to assess relationships between the social, personal and disease outcome measures. For further details of the statistical procedures see page 83.

### **3.2.0 PARTICIPANTS**

The groups of participants were recruited in different ways. A representative from the Tasmanian Lupus Society contacted members to explain the study. Of the 50 members, 34 volunteered to participate. This represents a response rate of 68 percent. The 34

volunteers met three or more ARA criteria and were diagnosed with SLE by either a specialist rheumatologist or general practitioner.

For individuals with MS, a written explanation of the study was forwarded by the Tasmanian Multiple Sclerosis Society only to members with a diagnosis confirmed by a neurologist. This was accompanied by a postcard asking about demographic information and a reply paid envelope. Of the 280 members sent information about the study, 141 volunteered to participate. This represents a response rate of about 50 percent. Since it was not possible to interview all the volunteers, a random sample of 40 individuals with MS were selected. Complete data sets were obtained from 37 individuals with MS, three were eliminated from analyses due to missing data.

The control group was selected from various community facilities including clubs and social organisations. Complete data sets were obtained from 38 controls, after three were eliminated with missing data.

Controls were selected so that the distribution of age, gender and socioeconomic status (SES) was approximately equal to the SLE and MS groups (Table 2). None of the groups differed on the demographic measures [age,  $F(2,105)=1.56$ ,  $p=.21$ ; SES,  $F(2,106)=1.21$ ,

$p=.30$ ; gender,  $X^2=1.64$ ,  $df=2$ ,  $p=.44$ ; marital status<sup>3</sup>,  $X^2=2.12$ ,  $df=2$ ,  $p=.35$ ].

Table 2: Demographic characteristics of participants.

	SLE	MS	CONTROLS
<hr/>			
GENDER			
males	6	7	11
females	28	30	27
AGE (years)			
mean	49.05	44.19	44.78
SD	16.24	8.19	12.14
MARITAL STATUS			
single	2	6	9
married/defacto/widowed	29	28	27
separated/divorced	3	3	2
DANIELS SCORE (OCCUPATIONAL SES; Daniels, 1983)			
mean	31.94	30.24	29.28
SD	7.48	4.97	8.82
<hr/>			

All participants gave written informed consent to take part in the study. A copy of the consent form appears in appendix one.

### 3.3.0 QUESTIONNAIRES

Only questionnaires derived from established psychological theories and with robust psychometric properties were used in the study. Copies of these questionnaires appear in appendix one. Since the

<sup>3</sup> The categories of single and separated/divorced were combined.



Sickness Impact Profile and the SCL-90-R have restricted availability, they do not appear in the appendix.

#### DEMOGRAPHIC AND DISEASE HISTORY QUESTIONNAIRE

The demographic questionnaire asks respondents about their age, sex and socioeconomic status as measured by the Daniels Score (Daniels, 1983). For individuals with SLE or MS, information about disease duration, medication and disease status was also collected.

#### SOCIAL RESOURCES

The social resources measures used in the present study (short-hand variable names in brackets) were daily hassles [HASSLES], uplifts [UPLIFTS], social network [NETWORK] and social support [SUPPORT].

#### DAILY HASSLES AND UPLIFTS SCALES [HASSLES & UPLIFTS]

Fifty-three items are rated for the amount of stress (hassles) and pleasure (uplifts) they have provided in the past 14 days (DeLongis, Folkman & Lazarus, 1988). Although separate scales have been devised to measure hassles and uplifts, it is recognised that a given event may have both qualities. Recent research suggests hassles and uplifts contribute differentially to disease outcomes, so there are included as separate dependent variables in the present study (Chamberlain & Zika, 1990).

#### NORBECK SOCIAL SUPPORT QUESTIONNAIRE [NETWORK]

The Norbeck Social Support Questionnaire was constructed to measure network size, functional support and social losses. In the present study, only the network size subscale is used since more robust social support measures are available. To complete the network measure, respondents list persons who are potential sources of support and specify the nominated person's relationship (e.g., family, coworker or practitioner). The network subscale has a test-retest reliability of .92 (Norbeck, Lindsay, & Carrieri, 1981).

#### INTERPERSONAL SUPPORT EVALUATION LIST [SUPPORT]

The Interpersonal Support Evaluation List (ISEL) comprises 40 statements concerning the availability of emotional, tangible, belonging and appraisal social support. The tangible subscale measures the availability of material aid; the appraisal subscale assesses the availability of someone with whom to discuss problems; the self-esteem subscale measures the availability of a positive comparison when comparing one's self with others; and the belonging subscale assesses the availability of people with whom one can do things. The true/false format of the ISEL is counterbalanced, with half the items inquiring about positive aspects of social support and the other half about negative aspects. Scores on the four ISEL subscales are summed to yield an estimate of total social support. Test-retest reliabilities range between .63 and .70 for the individual subscales and internal consistencies from .88 to .99. The ISEL has

documented discriminant and convergent validity (see Cohen, Mermelstein, Kamarck & Hoberman, 1985).

## **PERSONAL RESOURCES**

The personal resource measures adopted in the present study (short-hand variable names in brackets) were self-efficacy [EFFICACY], emotion-focused coping [EMOTION] and problem-focused coping [PROBLEM].

### **SELF-EFFICACY [EFFICACY]**

The 22-item Coppel (1980) scale has documented psychometric data including internal validity (.91), test-retest reliability (.86), convergent validity and factor structure. It measures behaviours related to self-efficacy including behavioural maintenance and outcome expectancies. It is a disposition measure and, therefore, equally applicable to SLE, MS and healthy people. Self-efficacy levels are reported as a single total score.

### **COPE [EMOTION & PROBLEM]**

The COPE consists of 53 items that were derived from Lazarus and Folkman's (1984) notion of emotional- and problem-focused coping (Carver, et al., 1989). It consists of 13 subscales, 10 of which represent three broad coping domains. These domains are active (or problem-focused), supportive and maladaptive (emotion-focused) coping and are not summed to yield a total COPE score. The remaining three scales measure coping responses that are neither

adaptive nor maladaptive and are not included in the domain totals or the present study. Individual COPE subscales have an internal consistency ranging from .62 to .92, a two week test-retest reliability of .61, as well as documented discriminant and convergent validity (Caver et al., 1989).

Since the COPE assesses both the situational and dispositional aspects of coping behaviour and is derived from the Folkman and Lazarus model, it was chosen for the present study. To be consistent with the Folkman and Lazarus model, only scores on the the emotion- and problem-focused domains were used in the analysis. The subscales that comprise problem-focused and emotion-focused coping domain scores appear in Table 3.

## **DISEASE OUTCOMES**

Outcome measures are the consequences of illness and include (short-hand variable names in brackets) psychological distress [DISTRESS], physical disability [PHYSICAL] and psychosocial impairment [DISRUPTION].

### **PSYCHOLOGICAL DISTRESS [DISTRESS]**

The revised 90 item Symptom Check-List (SCL-90-R) is a self-report measure of current psychological distress, consisting of 90 items, each rated on a five-point scale (Derogatis, 1983). The scale yields nine primary symptom dimensions and three global scores of distress (Table 4). The general symptom index (GSI) is considered the most

informative psychopathology measure and is adopted in the present study (Brophy, Norvell & Kiluk, 1988). The SCL-90-R has a flexible timeframe and in the present study psychological distress over the past 14 days is assessed. The psychometric properties of the SCL-90-R are robust and have been reviewed by Brophy and associates (Brophy et al., 1988). A standardised SCL-90-R score of 63 is used to diagnose clinical syndromes (Derogatis, 1977). The present study is concerned with general (rather than clinical) distress levels and adopts the GSI index rather than the cut-off criteria.

#### SICKNESS IMPACT PROFILE [PHYSICAL & DISRUPTION]

The SIP is a self-report measure of disability and psychosocial disruption associated with medical conditions. Respondents are asked to endorse only those items which describe their health over the past 14 days (Bergner, 1977). Items are weighted according to the severity of limitation on behaviour implied by each statement. The items cover a range of domains including sleep and rest, eating, work, home management, recreation and pastimes, ambulation, mobility, emotional behaviour, body care and movement, social interaction and communication. These subscales are summed to yield a measure of psychosocial and physical disability, as well as an overall sickness impact score.

The psychometric properties of the SIP are reviewed by Wilkin and associates (Wilkin, Hallam & Doggett, 1992) and include high internal consistency, test-retest reliability and inter-rater reliability.

Table 3: The problem-focused and emotion-focused COPE subscales.  
(After Carver et al., 1989)

---

*PROBLEM-FOCUSED COPING*

**ACTIVE COPING**

Taking action, exerting efforts, to remove or circumvent the stressor.

**PLANNING**

Thinking about how to confront the stressor, planning one's active coping efforts.

**SUPPRESSION OF COMPETING ACTIVITIES**

Suppressing one's attention to other activities in order to concentrate on coping with the stressor.

**POSITIVE REINTERPRETATION**

Making the best of a situation by viewing it in a more favourable light.

**RESTRAINT**

Holding back one's coping efforts until they are effective.

*EMOTION-FOCUSED COPING*

**ACCEPTANCE**

Accepting that the stressful event has occurred.

**FOCUS ON & VENTING EMOTIONS**

Increased awareness of emotional stress and a tendency to vent emotional distress.

**DENIAL**

Attempts to reject the reality of a stressful event.

**MENTAL DISENGAGEMENT**

Psychological disengagement from the goal with which the stressor is interfering, through daydreaming, sleep, or self-distraction.

---

Table 4: The SCL-90-R symptom dimensions  
and global distress indices.

---

**SOMATIZATION**

This dimension measures distress arising from bodily dysfunction.

**OBSESSIVE-COMPULSIVE**

Measures thoughts, impulses and actions that are experienced as unremitting and irresistible.

**INTERPERSONAL SENSITIVITY**

Focuses on feelings of personal inadequacy and inferiority.

**DEPRESSION**

Measures symptoms indicative of depression including dysphoric mood, withdrawal, loss of interest in usual activities and decreased energy levels.

**ANXIETY**

Assesses clinical signs of anxiety including nervousness, tension, panic attacks, feelings of terror and trembling.

**HOSTILITY**

Measures thoughts, feelings or actions that characterise the state of anger.

**PHOBIC ANXIETY**

This is a specific fear response to a person, place, object or situation which is characterised by irrational, avoidance or escape behaviour.

**PARANOID IDEATION**

Measures paranoid behaviours including protective thought, hostility, suspiciousness, grandiosity, centrality and delusions.

**PSYCHOTICISM**

This dimension measures behaviour indicative of schizophrenia and schizoid personality disorder. It assesses withdrawal, isolation, hallucinations and thought broad-casting.

**GLOBAL INDICES OF DISTRESS**

1. Global Severity Index (GSI)

This score is considered the best single index of global distress, it combines information on the number of symptoms and intensity of perceived distress.

2. Positive Symptom Distress Index (PSDI)

The PSDI is a measure of distress intensity corrected for the number of symptoms reported.

3. Positive Symptom Total (PST)

This assesses the number of symptoms reported independent of their severity.

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The SIP also demonstrates high correlations with physician rated disability. It has been used in over 40 studies of patients suffering from various illnesses including chronic pain, arthritis, cancer, angina, heart failure, fatigue and lung diseases (Wilkinet al., 1992).

Since the SIP assesses disability and psychosocial disruption resulting from illness, it is easily administered to both SLE and MS sufferers, as well as healthy controls. The present study uses physical disability and psychosocial disruption scores as distinct disease outcome measures. Examples of items comprising the physical SIP subscales appear in Table 5.

### **3.4.0 PROCEDURE**

Data were gathered by conducting personal interviews so as to establish rapport and to insure completion of the questionnaires. Since SLE and MS are uncommon medical conditions, it was necessary to travel throughout Tasmania to interview the volunteers. Interviews were conducted at sufferers' residences and at a convenient time convient to the volunteer. For 95 percent of individuals, two short interviews were arranged each lasting about 30-40 minutes. In the first interview rapport was established and several questionnaires completed. An explanation of the remaining questionnaires was then provided and individuals completed these over a 10-14 day period. During the second interview, questionnaires were scanned for their completeness and any questions volunteers raised answered.



Table 5: Examples of items comprising the physical disability and psychosocial disruption subscales.

-----	
PHYSICAL DISABILITY	
BODY CARE & MOVEMENT	
	I stand for only short periods of time.
	I do not maintain balance.
	I am in a restricted position all the time.
	I stay lying down most of the time.
MOBILITY	
	I am getting around only within one building.
	I stay home most of the time.
	I stay away from home for only brief periods of time.
	I am staying in bed more.
PSYCHOSOCIAL DISRUPTION	
COMMUNICATION	
	I am having trouble writing or typing
	I am understood with difficulty
	I do not speak clearly when I am under stress
EMOTIONAL BEHAVIOUR	
	I act nervous or restless
	I talk about the future in a hopeless way
	I get sudden frights
-----	

One SLE and 10 MS sufferers reported visual problems or motor difficulties. For these individuals several short visits were arranged over a two week period where questions were read and answers scribed.

Controls were screened for the presence of a chronic illness or chronic symptomatology. Individuals in the healthy comparison group received a financial reward for completing the questionnaires.

Questionnaires were scored according to their devised criteria and the data analysed using SPSS (version 4) and Statview III.

## Chapter Four

### Results

The design consisted of three groups matched for age and socioeconomic status. To control for Type I errors, group differences were first assessed by performing a one-way multivariate analysis of variance (MANOVA) on each group of dependent variables (social, personal and disease outcome measures). To elucidate differences found to be significant by MANOVA, one-way analyses of variance (ANOVA) and posthoc LSD tests were performed on each dependent variable to test differences between pairs of means. The LSD tests were interpreted as significant only if the preceding ANOVA was significant. Correlation matrices for the separate groups were also determined. The significant correlations are considered only briefly, as the purpose of the matrices is to explain the possible processes underlying group differences.

Stepwise multiple regression was used to determine which social and personal resource variables predicted disease outcomes and to establish the strength of any relationships. These analyses were performed separately for each group. An alpha level of .05 was adopted for all statistical tests.

#### **4.1.0 GROUP COMPARISONS**

Table 6 contains means and standard deviations for the social, personal and disease outcome measures. For the social measures the one-way MANOVA was statistically significant [Wilks Lambda=.81,  $F(8, 206) = 2.85, p = .0051$ ]. Group differences were present for the uplifts [ $F(2, 106) = 5.43, p = .0057$ ] and social support [ $F(2, 106) = 4.54, p = .0127$ ] scales. Differences were attributable to SLE sufferers reporting fewer

uplifts than healthy controls or MS sufferers and the chronic illness groups scoring lower on the social support scale (Table 7). No group differences were evident on the hassles or network scales.

For personal resource measures, the one-way MANOVA was also statistically significant [Wilks Lambda=.80,  $F(6, 208) = 3.94$ ,  $p=.0009$ ]. Group differences for the self-efficacy scale [ $F(2, 106) = 3.68$ ,  $p=.0283$ ] were due to healthy controls scoring higher than either the SLE or MS groups (Table 7). Differences on the emotion-focused coping scale [ $F(2, 106) = 5.89$ ,  $p=.0037$ ] were due to controls using fewer of these strategies than the SLE and MS groups (Table 7). There were no significant group differences on the problem-focused coping scale.

Expected differences on the disease outcome measures were also present [Wilks Lambda=.55,  $F(6, 208) = 12.22$ ,  $p=.0001$ ]. All groups differed statistically on the physical disability scale [ $F(2, 106) = 24.81$ ,  $p=.0001$ ] with the MS group reporting the highest and controls the lowest rates (Table 7). Significant differences on the psychosocial disruption measure [ $F(2, 106) = 17.69$ ,  $p=.0001$ ] arose from the SLE and MS groups scoring higher than healthy controls (Table 7). Finally, differences on the psychological distress measures [ $F(2, 106) = 21.13$ ,  $p=.0001$ ] arose from controls reporting lower levels than the chronic illness groups (Table 7).

Table 6: Mean and standard deviation scores for social, personal and disease outcome measures.

SCALES	SLE (n=34)	MS (n=37)	CONTROLS (n=38)
<hr/>			
<i>SOCIAL RESOURCES</i>			
HASSLES			
mean	25.02	26.89	32.28
SD	19.93	17.64	24.40
UPLIFTS			
mean	35.52	43.13	53.89
SD	22.13	20.27	28.06
NETWORK			
mean	14.44	12.59	13.07
SD	6.77	8.31	5.94
SUPPORT			
mean	29.02	29.78	33.86
SD	8.58	8.62	4.36
 <i>PERSONAL RESOURCES</i>			
EFFICACY			
mean	62.17	62.05	68.21
SD	11.18	12.97	9.06
PROBLEM*			
mean	67.97	61.99	62.73
SD	13.63	14.98	16.71
EMOTION*			
mean	55.56	56.12	47.58
SD	10.63	9.73	14.81
 <i>DISEASE OUTCOME MEASURES</i>			
DISTRESS			
mean	60.29	62.75	48.02
SD	10.23	9.95	11.20
DISABILITY			
mean	5.51	16.11	0.39
SD	8.73	14.61	1.03
DISRUPTION			
mean	12.93	17.32	1.52
SD	13.77	15.16	3.10

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PROBLEM\*= Problem-focused coping, EMOTION\*= emotion-focused coping

Table 7: Univariate comparisons and post hoc tests for social, personal and disease outcome measures for the control (C), SLE and MS groups.

SCALES	F	df	p	LSD*
<hr/>				
<i>SOCIAL RESOURCES</i>				
HASSLES	1.19	2,106	.3082	<u>C&gt;MS&gt;SLE</u>
UPLIFTS	5.43	2,106	.0057	<u>C&gt;MS&gt;SLE</u>
NETWORK	.64	2,106	.5281	<u>SLE &gt;C&gt;MS</u>
SUPPORT	4.55	2,106	.0127	<u>C&gt;MS&gt;SLE</u>
<i>PERSONAL RESOURCES</i>				
EFFICACY	3.69	2,106	.0283	<u>C&gt;SLE&gt;MS</u>
PROBLEM*	1.62	2,106	.2043	<u>SLE&gt;C&gt;MS</u>
EMOTION*	5.89	2,106	.0037	<u>MS&gt;SLE&gt;C</u>
<i>DISEASE OUTCOME MEASURES</i>				
DISTRESS	21.13	2,106	.0001	<u>MS&gt;SLE&gt;C</u>
DISABILITY	24.81	2,106	.0001	<u>MS&gt;SLE&gt;C</u>
DISRUPTION	17.69	2,106	.0001	<u>MS&gt;SLE&gt;C</u>

\*Groups with a common underline do not have significantly different means

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PROBLEM\*= Problem-focused coping, EMOTION\*= emotion-focused coping

## 4.2.0 RELATIONSHIPS BETWEEN THE VARIABLES

The correlation matrices for the separate groups are presented in Table 8, with statistically significant coefficients being indicated by bold type. More variables were significantly correlated for the control group and fewer for the SLE sufferers. The dissimilar correlation patterns are

considered further in the discussion as explanations for the group differences observed above.

#### 4.3.0 PREDICTING DISEASE OUTCOMES

Stepwise multiple regression was used to determine which social and personal resource variables predicted disease outcomes and to establish the strength of their relationships. An inspection of the correlations between variables (Table 8) suggests they interact differentially to influence disease outcomes for the different groups. Consequently stepwise regressions were performed separately for each group. An F-to-enter value of 4.17 was derived from the degrees-of-freedom for the smallest group [SLE sufferers  $df=(1,32)$ ].

##### DISTRESS

For the SLE group, social and personal resources were not correlated with psychological distress scores. In the stepwise analysis, however, hassles and uplifts predicted psychological distress scores (Table 9). At step one hassle scores were entered into the equation [ $R^2 = .18$ ,  $[F(1, 32) = 7.12, p < .05]$ ]. Uplifts were entered at step two [ $R^2 = .30$ ,  $[F(2, 31) = 6.51, p < .01]$ ]. No additional variables were entered into the equation. The standardised regression coefficients suggest decreasing uplifts and increasing hassles are associated with elevated psychological distress.

For the MS group, none of the observed social and personal variables were significantly correlated with the measure of psychological distress (Table 9).



Table 8: Correlation matrices for SLE (n=34),  
MS (n=37) and control (n=38) groups\*.

SLE		A	B	C	D	E	F	G	H	I
HASSLES	A									
UPLIFTS	B	.07								
NETWORK	C	-.11	.11							
SUPPORT	D	<b>-.51</b>	.06	.23						
EFFICACY	E	-.11	.23	-.12	.30					
PROBLEM*	F	-.01	.27	-.00	.22	<b>.36</b>				
EMOTION*	G	.27	.00	-.25	-.14	.08	<b>.55</b>			
DISTRESS	H	<b>.43</b>	-.31	-.13	-.35	-.34	-.22	.22		
DISABILITY	I	.27	-.08	.11	-.26	-.13	.16	.04	.12	
DISRUPTION	I	<b>.69</b>	-.07	.01	<b>-.46</b>	-.26	-.07	.21	<b>.54</b>	<b>.62</b>
MS		A	B	C	D	E	F	G	H	I
HASSLES	A									
UPLIFTS	B	<b>.36</b>								
NETWORK	C	-.08	.23							
SUPPORT	D	-.17	<b>.36</b>	.26						
EFFICACY	E	-.09	.21	.17	<b>.48</b>					
PROBLEM*	F	-.13	<b>.51</b>	.26	<b>.44</b>	<b>.61</b>				
EMOTION*	G	.12	.28	.26	.25	.10	<b>.49</b>			
DISTRESS	H	.23	.22	.10	-.28	-.29	-.02	-.03		
DISABILITY	I	-.06	-.17	-.15	<b>-.56</b>	-.13	-.13	-.26	.33	
DISRUPTION	I	.19	-.19	.17	<b>-.54</b>	<b>-.53</b>	-.30	.06	<b>.64</b>	<b>.46</b>
CONTROLS		A	B	C	D	E	F	G	H	I
HASSLES	A									
UPLIFTS	B	<b>.37</b>								
NETWORK	C	-.19	.30							
SUPPORT	D	-.30	.15	<b>.42</b>						
EFFICACY	E	-.16	.00	.10	<b>.41</b>					
PROBLEM*	F	.28	<b>.75</b>	.23	.02	-.01				
EMOTION*	G	<b>.54</b>	<b>.61</b>	-.07	<b>-.34</b>	<b>-.36</b>	<b>.69</b>			
DISTRESS	H	.31	.10	<b>-.48</b>	<b>-.54</b>	-.41	.21	<b>.60</b>		
DISABILITY	I	.05	-.14	-.24	-.08	-.08	.02	-.12	-.04	
DISRUPTION	J	<b>.46</b>	.10	-.10	-.21	-.15	<b>.36</b>	<b>.33</b>	<b>.43</b>	.23

\* Statistically significant correlations ( $p < .05$ ) are in bold type  
 PROBLEM\* = Problem-focused coping, EMOTION\* = emotion-focused coping resource measures

Table 9: Summary of the stepwise regression for variables predicting psychological distress.

	Variable	B	SE B	$\beta$
SLE	Hassles	.23	.08	.45
$R^2=.30$	Uplifts	-.15	.07	-.34
MS	No significant predictors			
CONTROLS	Emotion*	.43	.09	.57
$R^2=.56$	Network	-.83	.21	-.44
Emotion*=emotion-focused coping				

For control controls, emotion-focused coping, social support and network size were correlated with psychological distress (Table 8). In the stepwise analysis, emotion-focused coping was first entered into the equation [ $R^2 = .36$ ,  $[F(1, 36) = 20.56, p < .01]$  (Table 9). At step two, network size was entered [ $R^2 = .56$ ,  $[F(2, 35) = 21.98, p < .01]$ . After step two, the remaining variables did not have an F-value greater than 4.17 and were not entered into the equation. The standardised regression coefficients indicated that a small social network and more emotion-focused coping was associated with higher psychological distress scores for control controls.

#### PHYSICAL DISABILITY

For the SLE group, social and personal resource measures were not correlated and did not predict physical disability levels. For the MS

group, however, social support was significantly correlated with and predicted physical disability levels [ $R^2 = .32$ ,  $[F(1, 35) = 16.18, p < .01]$ . From the standardised regression coefficient (Table 10) more physical disability was associated with less social support. For control group, social and personal resource measures were not correlated and did not predict physical disability levels.

Table 10: Summary of the stepwise regression for variables predicting physical disability levels.

	Variable	<u>B</u>	<u>SE B</u>	<u><math>\beta</math></u>
SLE	No significant predictors			
MS <u><math>R^2 = .32</math></u>	Social support	-.95	.24	-.56
CONTROLS	No significant predictors			

#### PSYCHOSOCIAL DISRUPTION

For the SLE group, disruption was correlated with hassles and social support. However, hassle levels were the only significant predictor of psychosocial disruption for SLE sufferers [ $R^2 = .48$ ,  $[F(1, 32) = 28.99, p < .01]$  (Table 11). Higher hassle levels were associated with more psychosocial disruption.

Social support and self-efficacy were significantly correlated with disability levels for MS volunteers. These, in addition to network size, predicted psychosocial disruption levels (Table 11). Social support was entered at step one [ $R^2 = .29$ ,  $[F(1, 35) = 14.06, p < .01]$ , network size at step

two [ $R^2 = .39$ ,  $F(2, 34) = 10.81, p < .01$ ] and self-efficacy levels at step three [ $R^2 = .50$ ,  $F(3, 33) = 10.84, p < .01$ ]. Higher psychosocial disruption levels were associated with less social support and self-efficacy, as well as a larger network size.

Table 11: Summary of the stepwise regression for variables predicting psychosocial disruption.

	Variable	<u>B</u>	<u>SE B</u>	<u>β</u>
SLE <u><math>R^2 = .48</math></u>	Hassles	.48	.08	.68
MS <u><math>R^2 = .50</math></u>	Support	-.79	.25	-.45
	Network	.64	.23	.35
	Efficacy	-.44	.16	-.37
CONTROLS <u><math>R^2 = .21</math></u>	Hassles	.06	.02	.46

For healthy controls, emotion- and problem-focused coping were correlated with disruption scores for healthy controls. Nevertheless, hassle levels were the only significant predictor of psychosocial disruption for the control group [ $R^2 = .21$ ,  $F(1, 36) = 9.82, p < .01$ ]. Where a moderate increase in hassles were associated with more psychosocial disruption. This finding is, however, unreliable given the large proportion of participants scoring zero disruption.

## Chapter Five

### Discussion

### **5.1.0 OVERVIEW**

The discussion parallels the results section, commenting first on the group differences for social, personal and disease outcome measures. For several measures, the limited research with SLE sufferers makes the findings difficult to interpret theoretically. By comparing correlation matrices (Table 8), however, some provisional hypotheses about the processes underlying group differences can be generated. The second section discusses the significance of social and personal variables in predicting disease outcome measures.

### **5.2.0 GROUP COMPARISONS**

#### **SOCIAL RESOURCES**

The MANOVA for social resource measures was statistically significant. Group differences were present for the uplifts and social support scales, but not of the hassle or network measures.

#### **HASSLES**

Although significant group differences were not evident on the hassles (stress) measure, controls reported marginally higher rates than the illness groups. This finding is contrary to the prediction that stress levels would be substantially elevated in the illness groups and may be reconciled by examining the methodology of published research.

Published research implies that high stress precedes disease exacerbations, but does not consider whether levels differ from other chronic illnesses

or 'healthy' controls (e.g., Laing et al., 1984; Rimon & Kronqvist, 1988). These studies also use hospitalised samples and may, therefore, be biased towards individuals with few coping skills or a more serious illness. Furthermore, they neglect to consider possible confounding between symptomatology, recent changes in the availability of social support and stress (see Thoits, 1985 for a review).

There also may be behavioural explanations for the observed differences in reported stress levels. For example, SLE sufferers may implicitly or explicitly avoid stress as they are more susceptible to its deleterious effects. Alternatively, being chronically ill may limit the range of activities in which individual can participate and hence the potential for stress. Taking account of activity levels would thus be more appropriate for assessing the effects of stress.

The interaction of hassles with the other variables provides another explanation of the possible processes underlying group differences. For the SLE group, high stress and low social support lead to psychological distress and psychosocial disruption. This finding is consistent with the buffering hypothesis of social support and is widely supported by research (see Cohen & Wills, 1985 for a review). High stress levels were associated with more positive events for MS sufferers, but not with disease outcome measures. This suggests high activity levels are both pleasurable and stressful for MS sufferers but are less important to disease outcome. For

controls, high stress [and uplifts] in the presence of emotion-focused coping leads to psychosocial disruption.

Whereas studies suggest SLE sufferers experience elevated stress levels, this finding may result from methodological limitations such as a lack of comparisons groups, using seriously ill hospitalised sufferers and confounded stress assessments. The low stress levels also may result from behavioural factors such as leaving the work force or avoiding potentially stressful activities. Correlational data suggest stress in the absence of sufficient social support may lead to psychological distress in SLE sufferers.

#### UPLIFTS

Whereas SLE research uses daily hassle methodology, the contradistinction that uplifts (or positive events) are important predictors of disease outcome is not widely investigated. Using the latter measure, significant differences in the number of uplifts were evident with controls differing from the chronic illness groups.

This finding is consistent with the notion that chronic illness limits activity and hence access to pleasurable events. Cognitive styles that disregard the positive aspects of events or high psychological distress levels also may influence reporting of pleasurable events, with distressed individuals reporting fewer pleasurable events (Macgillivray & Baron, 1994).



The interaction of uplifts with other variables may also suggest reasons for group differences on the scale. Uplifts were not correlated with any social, personal or disease outcome measure for the SLE group. Thus, uplifts are less important to the well-being of SLE sufferers. For the MS group, uplifts are associated with more social support, problem-focused coping and stress. They were, however, unrelated to the disease outcome measures. Thus, for individuals with MS, activity levels may depend on the presence of social support which in turn promotes problem-focused coping. Uplifts were associated with more stress, problem- and emotion-focused coping for the control group. For controls, high activity levels promote both more positive and negative coping strategies.

#### SOCIAL NETWORKS AND SOCIAL SUPPORT

Although not statistically significant, SLE sufferers reported more social network members than either control or MS subjects. This finding is interesting as it suggests that there has been no decrease in network size in the SLE group over the course of the disease. When compared with the illness groups however, control subjects reported significantly more social support. Thus, despite embeddedness in a social network, chronic illness sufferers may receive less social support than healthy people. The correlation matrices may provide insight into how and why social relationship vary between the groups.

For healthy controls, social support is influenced differentially by social and personal resource variables. Social network size and social support

are highly correlated, suggesting embeddedness is associated with potentially more social support. High social support also is associated with greater self-efficacy and using less emotion-focused coping. Low social support and a small network are related to increased psychological distress, but not to the other disease outcome measures. Causal associations aside, the intercorrelations between variables suggest that low self-efficacy and emotion-focused coping in the presence of low social support are associated with less psychological distress in healthy people.

For the SLE group, social support is associated with different social and personal resource variables. Lower social support was associated with more stress and psychosocial disruption. That is, there was evidence for the buffering model of social support.

For MS sufferers, high social support was significantly associated with greater self-efficacy, problem-focused coping and more uplifts. The aforementioned correlations suggest that positive events are associated with greater self-efficacy which improves problem-focused coping and enhances social support levels. This hypothesis has some support from the notion that social support promotes coping assistance which in turn minimises negative disease outcomes (Thoits, 1986).

Another important implication from these post hoc analyses is that social support is differentially related to disease outcome measures. For control subjects, low social support was associated with more psychological

distress, but not the other indices of health status. This finding is unsurprising given that controls were selected for their absence of ill health. For SLE sufferers, high social support was associated with less psychosocial disruption, but not with psychological distress or disability. Thus, high social support may have a buffering role against psychosocial disruption. Equally low levels of social support may be a consequence of psychosocial disruption. For the MS group, low social support was associated with greater physical disability and psychosocial disruption and unrelated to psychological distress scores. It is possible that high disability mitigates social support levels which in turn lead to increases in psychosocial disruption (Brown et al., 1989).

Although it was theoretically possible to investigate the buffering hypothesis for social support in the present study, this is not recommended when data is cross-sectional (Felner et al., 1983; Thoits, 1985; Cohen & Wills, 1985; Gottlieb, 1988). Cross-sectionally, high stress levels have been a demonstrated consequence of low social support and low stress levels are correlated with high social support (Felner et al., 1983; Thoits, 1985; Cohen & Wills, 1985; Gottlieb, 1988). Thus, preexisting stress levels may be an outcome of existing social support. Furthermore, high stress levels may arise from a loss of social support, again confounding outcomes. The aforementioned difficulties make longitudinal data that controls for time-1 levels of stress and social support the minimal requirement for testing the buffering hypothesis (Felner et al., 1983; Thoits, 1985; Cohen et al., 1985; Cohen & Wills, 1985; Gottlieb, 1988; Brown

et al., 1989; Fitzpatrick et al., 1991).

## **PERSONAL RESOURCES**

The MANOVA for personal resource measures was statistically significant. Group differences were present for the self-efficacy and emotion-focused coping scale, but not of the problem-focused coping measure.

### **SELF-EFFICACY**

The prediction that self-efficacy levels would be significantly higher for controls than for either illness group was supported. The antecedents of lowered levels are not evident from this study, but it is likely that the unpredictable relapsing and remitting course of SLE may lower self-efficacy perceptions as individuals believe they lack the skills to alleviate symptomatology.

The group differences may again be clarified by considering the association of efficacy with other variables. For both chronic illness groups, problem-focused coping was significant related to self-efficacy. This finding also has been reported with RA sufferers (Schiaffino et al., 1991). For SLE sufferers, self-efficacy was not significantly related to any other social, personal or outcome variables. For individuals with MS, high self-efficacy was related to low disability which is consistent with findings from other studies (O'Leary et al., 1988; Schiaffino et al., 1991). High self-efficacy also was significantly related to higher social support which is again consistent with the findings from other studies (O'Leary et al., 1988).

Thus, for SLE sufferers high self-efficacy may promote problem-focused coping or vice versa, and for MS sufferers this relationship is further mediated by high social support and disability levels.

For controls high self-efficacy was correlated with high social support. It also was correlated with using fewer emotion-focused coping strategies, but not with problem-focused coping. Thus, self-efficacy may promote social support and limit unproductive emotion-focused coping.

#### PROBLEM- AND EMOTION-FOCUSED COPING

No significant group differences were present on the problem-focused coping measure. Significant group differences on the emotion-focused coping measure were evident. The chronic illness group used more emotion-focused strategies than control subjects. Thus, chronically ill individuals use problem-focused strategies as often as healthy controls. They also use more emotion-focused strategies that are less advantageous to overall well-being. This finding is not widely reported, as most researchers classify individuals as either emotion- or problem-focused copers to compare outcome measures (e.g., Brown et al., 1989; Revenson & Felton, 1989; Newman et al., 1990). This typology is not justified in the present study as emotion- and problem-focused coping were highly correlated in all the groups (Table 8).

Group differences in coping strategies also may be clarified by considering their association with other variables. For the chronic illness groups,

individuals using problem-focused coping strategies reported higher self-efficacy scores. No evidence was found for the previously reported finding that emotion-focused coping would be significantly associated with either low self-efficacy [or high disability] scores (Revenson & Felton, 1989). No other significant associations between coping and the other variables were evident for SLE sufferers. For the MS group, high scores on the problem-focused coping scale were associated with more positive events and social support. Furthermore, individuals with high scores on the emotion-focused coping scale reported less psychosocial disruption. Finally, in the chronic illness groups no support was found for problem-focused coping being associated with lower disability levels (Newman et al., 1990), emotion-focused coping being associated with more psychological distress (Brown et al., 1989) or problem-focused coping being associated with less psychological distress (Brown et al., 1989).

For controls, coping was complexly related to the other variables. Higher problem-focused coping scores were associated with more uplifts and psychosocial disruption. Higher emotion-focused coping scores were associated with more hassles, uplifts, psychological distress and psychosocial disruption. Emotion-focused coping also was associated with less social support and lower self-efficacy scores. Thus, it appears that when coping is important, both emotion- and problem-focused coping strategies are employed. Individuals with higher emotion-focused scores, however, experience lower social support and lower self-efficacy

levels. It is implicit from the correlation matrix for controls that emotion-focused coping may be adaptive at low levels, but detrimental to personal or social resources when used extensively. The COPE scale may also have multicollinearity problems, with emotion-focused items overlapping with measures of social support, psychological distress and psychosocial disruption.

### **DISEASE OUTCOME MEASURES**

The MANOVA for disease outcome measures was statistically significant. Group differences were present for the psychological distress, disability and psychosocial disruption measures.

### **PSYCHOLOGICAL DISTRESS**

The prediction that chronic illness would be associated with more psychological distress was supported, with distress levels being comparable between SLE and MS sufferers, who both differed significantly from controls.

For the SLE group, psychological distress was associated with more hassles and psychosocial disruption. These findings have been widely reported (e.g. Otto & Mackay, 1967; Laing et al., 1984; Rimon & Kronqvist, 1988; Wekking et al., 1991) in SLE sufferers. Curiously, other social and personal resources variables were not associated with psychological distress scores suggesting specific etiological factors in SLE sufferers. For the MS group, higher psychological distress was related to more psychosocial disruption.

No other significant correlations were evident for MS sufferers. For controls, high psychological distress was associated with a smaller social network and less social support. These findings have been widely reported in studies with healthy people (see Cohen & Wills, 1985 for a review). Psychological distress also was associated with using more emotion-focused coping and greater psychosocial disruption.

#### DISABILITY AND PSYCHOSOCIAL DISRUPTION

The groups were significantly different on the disability measure with MS sufferers scoring higher than SLE, who in turn differed from controls. This finding is unremarkable as controls subjects were selected for the absence of illness. The higher score of MS sufferers was also expected, as this group experiences more mobility problems than SLE sufferers.

Psychosocial disruption scores for the chronic illness groups also differed significantly from controls. With scores of SLE and MS sufferers being comparable due to symptomatology causing disruption independently of its etiology. The lower scores for control subjects are unremarkable as this group were free from ill health.

The Disease Exacerbation Model predicted a deterioration in all social and personal resources as well as in disease outcomes. While significant decrements were observed on all disease outcome measures when comparing the chronic illness and control groups, there were some social and personal resources not apparently influenced by the course of disease.



In particular, there was no significant difference between the chronic illness and control groups on measures of hassles, network size or problem-focused coping. These findings provide a significant qualification in refining the scope of the Disease Exacerbation Model for chronic illness.

### **5.3.0 PREDICTORS OF DISEASE OUTCOME**

While group differences may be partially explained by the differential interaction of variables, their importance in disease outcome was established through stepwise multiple regression analyses. The discussion again parallels the results section format.

#### **PSYCHOLOGICAL DISTRESS**

For the SLE group, hassles in the absence of uplifts predicted psychological distress and accounted for 30 percent of the variance. Uplifts appear to have a buffering or compensatory effect in that they were not significantly associated with psychological distress in the correlation matrix (Table 8). It is consistent with studies using 'healthy people' and the theoretical underpinnings of hassle and uplift research proposed by Delongis and associates (1988). That is, high stress in the absence of counterbalancing uplifts (positive events) leads to psychological states such as anxiety and depression as well as physical symptomatology (Delongis et al., 1988).

There were no predictors for psychological distress in the stepwise model for MS sufferers. This finding is consistent with correlational data. That

is, psychological distress was not significantly correlated with either social or personal resource variables. Thus psychological distress in MS sufferers is influenced by variables not included in the current model and has a different etiology than for the SLE group.

For the control group, emotion-focused coping and a small social network predicted psychological distress. These variables accounted for 56 percent of the observed variance for psychological distress scores. This etiology is different to the hassles and uplifts model observed in SLE sufferers, and implies a main-effect for social networks in the presence of poor coping (Cohen & Wills, 1985).

### **PHYSICAL DISABILITY**

For the SLE and control groups, neither social nor personal resources predicted disability scores. Since controls were selected for an absence of illness and hence disability, the null relationship is not unexpected. The lack of a significant relationship for SLE sufferers suggests that disability levels are not mediated by social and personal resources for SLE sufferers. For individuals with MS, however, insufficient social support predicted higher disability scores and accounted for 32 percent of the variance. This suggests that social support is important in assisting individuals to overcome mobility and related problems (Wineman, 1990).

### **PSYCHOSOCIAL DISRUPTION**

For controls and SLE sufferers, high hassles were associated with more

psychosocial disruption. For the SLE group, 48 percent of psychosocial disruption was attribute to hassles and is consistent with research findings (e.g. Otto & Mackay, 1967; Laing et al., 1984). For the MS group, 50 percent of psychosocial disruption was attributable to low social support in the presence of a large social network and poor self-efficacy.

#### **5.4.0 SUMMARY AND CONCLUSIONS**

This study examined the social, personal and disease outcome differences between SLE, MS and healthy control subjects. This comparative design provided partial support for the Disease Exacerbation Model of chronic illness. It demonstrated that individuals with SLE experienced similar problems to MS sufferers (with the exception that MS sufferers reported significantly more disability) and that both illness groups differed from healthy controls.

Both chronic illness groups reported less social support, more emotion-focused coping and lower self-efficacy. In addition, the chronic illness groups reported more psychological distress, disability and psychosocial disruption. No significant differences were, however, found between the chronic illness groups and healthy controls on the hassles, network size, or problem-focused coping measures. Some social and personal resources of ill people, therefore, remained intact despite suffering from a chronic disease.

The correlation matrices demonstrated that social, personal and disease outcome measures interact differentially to influence group differences. These interactions are, however, best investigated longitudinally controlling for time-1 levels of the variables. For example, the buffering hypothesis could not be adequately investigated with cross-sectional data as high stress has been demonstrated to be a function of low social support levels (Thoits, 1985; Cohen & Wills, 1985).

For the SLE group, hassles appear to be the most important moderator of disease outcome, but may be buffered by uplifts. Social support also was significantly related to psychosocial disruption and may be a mitigating factor. For MS sufferers, social support, networks and self-efficacy are the important social and personal resource variables mediating disease outcomes. While useful for comparison purposes, the regression findings are less unreliable for controls, as these individuals were free from chronic illness.

There are several methodological problems in this present study that limit the generalisation of findings. Individual matching was not possible in the present study because of limitations of available participants in the chronic illness groups. Furthermore, other social and personal resources not included in the present study may also be important mediators of disease outcomes. These include social resources such as negative social support transactions, as well as the personal resources such as cognitive styles and control. Finally, some of the hypotheses of interest in studying

the course of chronic illness are not adequately tested in a cross-sectional designs. For example, the buffering hypothesis for social support requires longitudinal data for adequate assessment. Despite these limitations, the present study has contributed to the identification of social and personal resources impacted upon by the process of two chronic diseases. It also has identified which social and personal resources potentially act as mediators in the disease process and has implications for intervention studies that may influence the course of chronic illness.

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## APPENDIX ONE QUESTIONNAIRES

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Social and Personal Resources as Mediators of Disease Outcome in  
Chronic Illness and Healthy People

**CONSENT FORM**

You are probably aware that having systemic lupus erythematosus (SLE)/multiple sclerosis (MS) may effect the quality of your life. Disruptions to life style could arise from disability, pain, restricted social contacts, your fatigue or medication. These stresses may make it more difficult to cope, lead to anxiety or depression and prolong exacerbations.

Research is currently underway investigating how SLE/MS effects the quality of your life. The study is open to people with SLE/MS throughout Tasmania, irrespective of the severity of your condition or where you live. The study is currently underway and involves two short interviews and some questionnaires to complete in your own time. A research assistant will visit you at home and help you to complete the questionnaires. If this is inconvenient other arrangements can be made. All information you provide will be kept confidential and only group data will be reported in scientific publications. You may withdraw from the study at any time, if you wish to do so.

If you would like more information you can contact Helen Hornsby Ph. (002) 202889 during office hours or the SLE/MS Society.

I have read and understand the consent form

Name\_\_\_\_\_

Address\_\_\_\_\_

Signature\_\_\_\_\_

Date\_\_\_\_\_

Witness\_\_\_\_\_

## REVISED DIAGNOSTIC CRITERIA FOR SYSTEMIC LUPUS ERYTHEMATOSUS

ed below is the diagnostic criteria for systemic lupus erythematosus. Please  
endorse the symptomatology your patient suffers, by ticking the appropriate box [ ☐ ].  
our patient is not a systemic lupus erythematosus sufferer, but fits some of the  
ow criteria, endorse the appropriate symptomatology. Over the page is a place for  
to specify your patient's diagnosis.

Criterion	Definition
[ <input type="checkbox"/> ] Malar Rash	Fixed erythema, flat or raised, over the malar eminences, tending to spare the nasolabial folds.
[ <input type="checkbox"/> ] Discoid Lupus	Erythematosus raised patches with adherent keratotic scaling and follicular plugging; atrophic scarring may occur in older lesions.
[ <input type="checkbox"/> ] Photosensitivity	Skin rash as a result of unusual reaction to sunlight, by patient history or physician observation.
[ <input type="checkbox"/> ] Oral ulcers	Oral or nasopharyngeal ulceration, usually painless, observed by physician.
[ <input type="checkbox"/> ] Arthritis	Nonerosive arthritis involving two or more peripheral joints, characterised by tenderness, swelling, or effusion.
[ <input type="checkbox"/> ] Serositis	a. Pleuritis- convincing history of pleuritic pain or rub heard by a physician or evidence of pleural effusion <b>OR</b> b. Pericarditis- documented by ECG or rub or evidence of pericardial effusion.

- 
- |       |                      |  |
|-------|----------------------|--|
| [   ] | Renal Disorder       | <ul style="list-style-type: none"><li>a. Persistent proteinuria greater than 0.5g/day or greater than 3+ if quantitation not performed<br/><b>OR</b></li><li>b. Cellular casts- may be red cell, hemoglobin, granular, tubular, or mixed.</li></ul>  |
| [   ] | Neurologic Disorder  | <ul style="list-style-type: none"><li>a. Seizures- in the absence of offending drugs or known metabolic derangements; e.g., uremia, ketoacidosis, or electrolyte imbalance<br/><b>OR</b></li><li>b. Psychosis- in the absence of offending drugs or known metabolic derangements; eg., uremia, ketoacidosis, or electrolyte imbalance.</li></ul>   |
| [   ] | Hematologic Disorder | <ul style="list-style-type: none"><li>a. Hemolytic anemia- with reticulocytosis<br/><b>OR</b></li><li>b. Leukopenia- less than 4000/mm<sup>3</sup> total on two or more occasions<br/><b>OR</b></li><li>c. Lymphopenia- less than 1500/mm<sup>3</sup> on two or more occasions<br/><b>OR</b></li><li>d. Thrombocytopenia- less than 100,000/mm<sup>3</sup> in the absence of offending drugs.</li></ul>  |
| [   ] | Immunologic Disorder | <ul style="list-style-type: none"><li>a. Positive LE cell preparation<br/><b>OR</b></li><li>b. Anti-DNA: antibody to native DNA in abnormal titer<br/><b>OR</b></li><li>c. Anti-Sm: presence of antibody to S m nuclear antigen<br/><b>OR</b></li><li>d. False-positive serologic test for syphilis known to be positive for at least 6 months and confirmed by <i>Treponema pallidum</i> immobilization or fluorescent treponemal antibody absorption test.</li></ul> |
| [   ] | Antinuclear Antibody | <p>An abnormal titer of antinuclear antibody by immunofluorescence or an equivalent assay at any point in time and in the absence of drugs known to be associated with "drug-induced lupus" syndrome.</p>  |
-

## DEMOGRAPHIC INFORMATION

Gender:      Female    ☐      Male      ☐      Age \_\_\_\_\_

Marital Status:                      single              ☐

   married              ☐

   separated              ☐

   divorced              ☐

   widowed              ☐

Do you live (tick as many as apply):

   on your own              ☐

   with your spouse partner              ☐

   with friends              ☐

   with brothers and/or sisters              ☐

   with parents              ☐

   with your children              ☐

   other, specify \_\_\_\_\_

Occupation \_\_\_\_\_

If a dependent, breadwinner's occupation \_\_\_\_\_

If a pensioner or unemployed, previous occupation \_\_\_\_\_

How old were you when diagnosed? \_\_\_\_\_

How long **before** diagnosis did you suffer from lupus/MS? \_\_\_\_\_

List any medication(s) prescribed to relieve your symptoms over the **past 6 months**. Include the **dosage level, frequency of ingestion, and how long** you have been taking each medication.

a. medication \_\_\_\_\_

dosage \_\_\_\_\_

frequency \_\_\_\_\_

duration \_\_\_\_\_

b. medication \_\_\_\_\_

dosage \_\_\_\_\_

frequency \_\_\_\_\_

duration \_\_\_\_\_

c. medication \_\_\_\_\_

dosage \_\_\_\_\_

frequency \_\_\_\_\_

duration \_\_\_\_\_

d. medication \_\_\_\_\_

dosage \_\_\_\_\_

frequency \_\_\_\_\_

duration \_\_\_\_\_

To what extent do the following medication **side effects** interfere with your daily functioning (e.g. ability to do housework, work or socialise).

a. increased appetite or weight gain

no disruption | severe disruption

b. depression

no disruption | severe disruption

c. stomach irritations

no disruption | severe disruption

d. fluid retention or moon face

no disruption

severe disruption

e. dizziness

no disruption

severe disruption

f. slow healing injuries

no disruption

severe disruption

g. being easily bruised

no disruption

severe disruption

Lupus/MS is characterised by periods of flare and remission. A **flare** occurs when your symptoms increase in intensity, possibly causing you considerable discomfort and distress. **Remission** occurs when there is an interval or break in the intensity of your symptoms, your discomfort is usually reduced.

What is your **current** illness condition?

flare [ ]

remission [ ]

early stage of a flare [ ]

late stage of a flare [ ]

never experienced a flare [ ]

"under control" (i.e., medication  
is preventing a flare) [ ]

not sure [ ]

About how long have you experienced your **current** (as above) illness condition?

---

## INSTRUCTIONS

HASSLES are irritants things that annoy or bother you; they can make you upset or angry. UPLIFTS are events that make you feel good; they can make you joyful, glad, or satisfied. Some hassles and uplifts occur on a fairly regular basis and others are relatively rare. Some have only a slight effect, others have a strong effect.

This questionnaire lists things that can be hassles and uplifts in day-to-day life. You will find that during the course of a day some of these things will have been hassles for you and some will have been only an uplift. *Others will have been both a hassle AND an uplift.*

**DIRECTIONS:** Please think about how much of a hassle and how much of an uplift each item was for you today. Please indicate on the left-hand side of the page (under "HASSLES") how much of a hassle the item was by circling the appropriate number. Then indicate on the right-hand side of the page (under "UPLIFTS") how much of an uplift it was for you by circling the appropriate number.

Remember, circle one number of the left-hand side of the page and one number on the right-hand side of the page for each item.

**HASSLES AND UPLIFT SCALE**

How much of a hassle was  
this item for you today?

**HASSLES**

0 = *None or not applicable*

1 = *Somewhat*

2 = *Quite a bit*

3 = *A great deal*

How much of an uplift was  
this item for you today?

**UPLIFTS**

*None or not applicable* = 0

*Somewhat* = 1

*Quite a bit* = 2

*A great deal* = 3

0	1	2	3	1. Your child(ren)	0	1	2	3
0	1	2	3	2. Your parents or parents-in-law	0	1	2	3
0	1	2	3	3. Other relative(s)	0	1	2	3
0	1	2	3	4. Your spouse	0	1	2	3
0	1	2	3	5. Time spent with your family	0	1	2	3
0	1	2	3	6. Health or well-being of a family member	0	1	2	3
0	1	2	3	7. Sex	0	1	2	3
0	1	2	3	8. Intimacy	0	1	2	3
0	1	2	3	9. Family related obligations	0	1	2	3
0	1	2	3	10. Your friend(s)	0	1	2	3
0	1	2	3	11. Fellow workers	0	1	2	3
0	1	2	3	12. Clients, customers, patients, etc.	0	1	2	3
0	1	2	3	13. Your supervisor or employer	0	1	2	3
0	1	2	3	14. The nature of your work	0	1	2	3
0	1	2	3	15. Your work load	0	1	2	3
0	1	2	3	16. Your job security	0	1	2	3
0	1	2	3	17. Meeting deadlines or goals on the job	0	1	2	3



HASSLES				HASSLES	UPLIFTS			
0 = <i>None or not applicable</i>					<i>None or not applicable</i> = 0			
1 = <i>Somewhat</i>					<i>Somewhat</i> = 1			
2 = <i>Quite a bit</i>					<i>Quite a bit</i> = 2			
3 = <i>A great deal</i>					<i>A great deal</i> = 3			
0	1	2	3	18. Enough money for necessities (e.g., food, clothing, housing, health care, taxes, insurance)	0	1	2	3
0	1	2	3	19. Enough money for education	0	1	2	3
0	1	2	3	20. Enough money for emergencies	0	1	2	3
0	1	2	3	21. Enough money for extras (e.g., entertainment, recreation, vacations)	0	1	2	3
0	1	2	3	22. Financial care for someone who doesn't live with you	0	1	2	3
0	1	2	3	23. Investments	0	1	2	3
0	1	2	3	24. Your smoking	0	1	2	3
0	1	2	3	25. Your drinking	0	1	2	3
0	1	2	3	26. Mood-altering drugs	0	1	2	3
0	1	2	3	27. Your physical appearance	0	1	2	3
0	1	2	3	28. Contraception	0	1	2	3
0	1	2	3	29. Exercise(s)	0	1	2	3
0	1	2	3	30. Your medical care	0	1	2	3
0	1	2	3	31. Your health	0	1	2	3
0	1	2	3	32. Your physical abilities	0	1	2	3
0	1	2	3	33. The weather	0	1	2	3
0	1	2	3	34. News events	0	1	2	3
0	1	2	3	35. Your environment (e.g., quality of air, noise level, greenery)	0	1	2	3

**HASSLES**0 = *None or not applicable*1 = *Somewhat*2 = *Quite a bit*3 = *A great deal***UPLIFTS***None or not applicable* = 0*Somewhat* = 1*Quite a bit* = 2*A great deal* = 3

0	1	2	3	36.	Political or social issues	0	1	2	3
0	1	2	3	37.	Your neighbourhood (e.g., neighbours, setting)	0	1	2	3
0	1	2	3	38.	Conserving (gas, electricity, water petrol etc.)	0	1	2	3
0	1	2	3	39.	Pets	0	1	2	3
0	1	2	3	40.	Cooking	0	1	2	3
0	1	2	3	41.	House work	0	1	2	3
0	1	2	3	42.	Home repairs	0	1	2	3
0	1	2	3	43.	Yardwork	0	1	2	3
0	1	2	3	44.	Car maintenance	0	1	2	3
0	1	2	3	45.	Taking care of paperwork (e.g., paying bills, filling of forms)	0	1	2	3
0	1	2	3	46.	Home entertainment (e.g., TV, music, reading)	0	1	2	3
0	1	2	3	47.	Amount of free time	0	1	2	3
0	1	2	3	48.	Recreation and entertainment outside home (e.g., movies, sports, eating out, walking)	0	1	2	3
0	1	2	3	49.	Eating (at home)	0	1	2	3
0	1	2	3	50.	Church or community organisations	0	1	2	3
0	1	2	3	51.	Legal matters	0	1	2	3
0	1	2	3	52.	Being organised	0	1	2	3
0	1	2	3	53.	Social commitments	0	1	2	3

## NSSQ

List each significant person in your life. Consider all the persons who have been important to you in the **past 6 months**. When listing individuals use only their first name or initials. Additionally, specify your relationship (e.g., spouse, family or relatives, friend, work associate, neighbour, general practitioner, counsellor etc.) with each of the nominated persons.

---

Name \_\_\_\_\_

Date \_\_\_\_\_

<b>First name or initials</b>	<b>Relationship</b>
1. _____	_____
2. _____	_____
3. _____	_____
4. _____	_____
5. _____	_____
6. _____	_____
7. _____	_____
8. _____	_____
9. _____	_____
10. _____	_____
11. _____	_____
12. _____	_____
13. _____	_____
14. _____	_____
15. _____	_____
16. _____	_____
17. _____	_____
18. _____	_____
19. _____	_____
20. _____	_____

**ISEL**

This scale is made up of a list of statements each of which may or may not be true about you. For each statement we would like you to circle TRUE if the statement is probably true about you or FALSE if the statement is probably not true about you.

You may find that many of the statements are neither clearly true or clearly false. In these cases, try to decide quickly whether TRUE or FALSE is most descriptive of you. Although some questions will be difficult to answer, it is important that you pick one alternative or the other. Remember to circle only one of the alternatives for each statement.

---

- |     |   |              |
|-----|---|--------------|
| 1.  | There is at least one person I know whose advice I really trust.                                    | True / False |
| 2.  | Most of my friends are more interesting than I am.  | True / False |
| 3.  | I feel that I'm on the fringe in my circle of friends.  | True / False |
| 4.  | I am more satisfied with my life than most people are with theirs.                                  | True / False |
| 5.  | I am able to do things as well as most other people.  | True / False |
| 6.  | When I feel lonely, there are several people I could call and talk to.                              | True / False |
| 7.  | There are very few people I trust to help solve my problems.  | True / False |
| 8.  | I have someone who takes pride in my accomplishments.   | True / False |
| 9.  | There is someone I can turn to for advice about handling hassles over household responsibilities.   | True / False |
| 10. | Most people I know think highly of me.  | True / False |
| 11. | If I got stranded ten kilometers out of town, there is someone I could call to come and get me.     | True / False |
| 12. | I think that my friends feel that I'm not very good at helping them solve problems.                 | True / False |
| 13. | If I needed some help in moving to a new home, I would have a hard time finding someone to help me. | True / False |

- |     |  |              |
|-----|--|--------------|
| 14. | If I decide on a Friday afternoon that I would like to go to a movie that evening, I could find someone to go with me. | True / False |
| 15. | I feel that there is no one with whom I can share my most private worries and fears.                                   | True / False |
| 16. | In general, people don't have much confidence in me.   | True / False |
| 17. | If a family crisis arose few of my friends would be able to give me good advice about handling it.                     | True / False |
| 18. | When I need suggestions for how to deal with a personal problem I know there is someone I can turn to.                 | True / False |
| 19. | There is no one I could call on if I needed to borrow a car for a few hours.   | True / False |
| 20. | There is really no one I can trust to give me good financial advice.   | True / False |
| 21. | I regularly meet or talk with members of my family or friends.   | True / False |
| 22. | If I need a quick emergency loan of \$100, there is someone I could get it from.                                       | True / False |
| 23. | There is someone who I feel comfortable going to for advice about sexual problems.                                     | True / False |
| 24. | In general people do not have much confidence in me.   | True / False |
| 25. | If I were sick, there would be almost no one I could find to help me with my daily chores.                             | True / False |
| 26. | If I needed a ride to the airport very early in the morning, I would have a hard time finding anyone to take me.       | True / False |
| 27. | No one I know would throw a birthday party for me.   | True / False |
| 28. | If for some reason I were put in jail, there is someone I could call to would bail me out.                             | True / False |
| 29. | Most of my friends are more successful at making changes in their lives than I am.                                     | True / False |

- |     |   |              |
|-----|---|--------------|
| 30. | Most people I know don't enjoy the same things that I do.   | True / False |
| 31. | If I were sick and needed someone to drive me to the doctor, I would have trouble finding someone.                                | True / False |
| 32. | There are several different people with whom I enjoy spending time.   | True / False |
| 33. | If I had to mail an important letter at the post office by 5:00 pm and couldn't make it, there is someone who could do it for me. | True / False |
| 34. | I don't often get invited to do things with others.   | True / False |
| 35. | There is someone I could turn to for advice about changing my job or finding a new one.   | True / False |
| 36. | If I wanted to have lunch with someone, I could easily find someone.  | True / False |
| 37. | I have a hard time keeping pace with my friends.  | True / False |
| 38. | If I had to go out of town for a few weeks, someone I know would look after my home (the plants, pets, yard, etc.).               | True / False |
| 39. | There is really no one who can give me objective feedback about how I'm handling my problems.                                     | True / False |
| 40. | I am closer to my friends than most other people.   | True / False |
-

*Read each statement carefully. Circle the number that best describes how you feel. There are no right or wrong answers.*

	Not at all like me	A little like me	Somewhat like me	Very much like me
1. Once I know what I need to do, I can do it.	1	2	3	4
2. In a new situation I expect I can handle things.	1	2	3	4
3. I am a confident person.	1	2	3	4
4. I am not very effective in solving problems.	1	2	3	4
5. When I'm stressed, I can count on myself to cope successfully.	1	2	3	4
6. I am not a self-assured person.	1	2	3	4
7. I have control over my reactions to stress.	1	2	3	4
8. I can usually get what I want.	1	2	3	4
9. I rely on my inner strength to deal with problems.	1	2	3	4
10. The good things that happen to me are largely my own doing.	1	2	3	4
11. I'm proud of myself.	1	2	3	4
12. I do not have a high opinion of my abilities.	1	2	3	4

	Not at all like me	A little like me	Somewhat like me	Very much like me
13. I wish I had more confidence in my ability to succeed in life.	1	2	3	4
14. People know they can expect a lot from me.	1	2	3	4
15. I believe I use my skills to their best advantage.	1	2	3	4
16. I am responsible for the ways I have grown as a person.	1	2	3	4
17. I can influence the people in my life.	1	2	3	4
18. I make my interactions with people end up the way I expect them to.	1	2	3	4
19. I am quick to learn new things about ways to deal with problems.	1	2	3	4
20. I am not afraid to make mistakes.	1	2	3	4
21. I know what people expect from me.	1	2	3	4
22. I question my abilities in difficult situations.	1	2	3	4



1 = I usually **don't** do this **at all**  
 2 = I usually do this a **little bit**  
 3 = I usually do this a **medium amount**  
 4 = I usually do this a **lot**

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1. I try to grow as a person as a result of the experience	1	2	3	4
2. I turn to work or other substitute activities to take my mind off things.	1	2	3	4
3. I get upset and let my emotions out.	1	2	3	4
4. I try to get advice from someone about what to do.	1	2	3	4
5. I concentrate my efforts on doing something about it.	1	2	3	4
6. I say to myself "this isn't real."	1	2	3	4
7. I put my trust in God.	1	2	3	4
8. I laugh about the situation	1	2	3	4
9. I admit to myself that I can't deal with it, and quit quickly.	1	2	3	4
10. I restrain myself from doing anything too quickly.	1	2	3	4
11. I discuss my feelings with someone.	1	2	3	4
12. I use alcohol or drugs to make myself feel better.	1	2	3	4
13. I get used to the idea that it happened.	1	2	3	4
14. I talk to someone to find out more about the situation.	1	2	3	4
15. I keep myself from getting distracted by other thoughts or activities.	1	2	3	4
16. I daydream about things other than this.	1	2	3	4
17. I get upset, and am really aware of it.	1	2	3	4
18. I seek God's help.	1	2	3	4
19. I make a plan of action.	1	2	3	4
20. I make jokes about it.	1	2	3	4

- |   |   |   |   |   |
|---|---|---|---|---|
| 21. I accept that this has happened and that it can't be changed.                           | 1 | 2 | 3 | 4 |
| 22. I hold off doing anything about it until the situation permits.                         | 1 | 2 | 3 | 4 |
| 23. I try to get emotional support from friends or relatives.                               | 1 | 2 | 3 | 4 |
| 24. I just give up trying to reach my goal.   | 1 | 2 | 3 | 4 |
| 25. I take additional action to try to get rid of the problem.                              | 1 | 2 | 3 | 4 |
| 26. I try to lose myself for a while by drinking alcohol or taking drugs.                   | 1 | 2 | 3 | 4 |
| 27. I refuse to believe it has happened.  | 1 | 2 | 3 | 4 |
| 28. I let my feelings out.  | 1 | 2 | 3 | 4 |
| 29. I try to see it in a different light, to make it seem more positive.                    | 1 | 2 | 3 | 4 |
| 30. I talk to someone who could do something concrete about the problem.                    | 1 | 2 | 3 | 4 |
| 31. I sleep more than usual.  | 1 | 2 | 3 | 4 |
| 32. I try to come up with a strategy about what to do.                                      | 1 | 2 | 3 | 4 |
| 33. I focus on dealing with this problem, and if necessary let other things slide a little. | 1 | 2 | 3 | 4 |
| 34. I get sympathy and understanding from someone.  | 1 | 2 | 3 | 4 |
| 35. I drink alcohol or take drugs, in order to think about it less.                         | 1 | 2 | 3 | 4 |
| 36. I kid around about it.  | 1 | 2 | 3 | 4 |
| 37. I give up the attempt to get what I want.   | 1 | 2 | 3 | 4 |
| 38. I look for something good in what is happening.   | 1 | 2 | 3 | 4 |
| 39. I think about how I might best handle the problem.                                      | 1 | 2 | 3 | 4 |
| 40. I pretend that it hasn't really happened.   | 1 | 2 | 3 | 4 |
| 41. I make sure not to make matters worse by acting too soon.                               | 1 | 2 | 3 | 4 |

- |   |   |   |   |   |
|---|---|---|---|---|
| 42. I try hard to prevent other things from interfering with my efforts at dealing with this. | 1 | 2 | 3 | 4 |
| 43. I go to movies or watch TV, to think about it less.                                       | 1 | 2 | 3 | 4 |
| 44. I accept the reality of the fact that it happened.  | 1 | 2 | 3 | 4 |
| 45. I ask people who have had similar experiences what they did.                              | 1 | 2 | 3 | 4 |
| 46. I feel a lot of emotional dsitress and I find myself expressing those feelings alot.      | 1 | 2 | 3 | 4 |
| 47. I take direct action to get around the problem.   | 1 | 2 | 3 | 4 |
| 48. I try to find comfort in my religion.   | 1 | 2 | 3 | 4 |
| 49. I force myself to wait for the right time to do something.                                | 1 | 2 | 3 | 4 |
| 50. I make fuin of the situation.   | 1 | 2 | 3 | 4 |
| 51. I reduce the amount of effort I'm putting into solving the problem.                       | 1 | 2 | 3 | 4 |
| 52. I talk to someone about how I feel.   | 1 | 2 | 3 | 4 |
| 53. I use alcohol or drugs to help me get hrough it.  | 1 | 2 | 3 | 4 |
| 54. I learn to live with it.  | 1 | 2 | 3 | 4 |
| 55. I put aside other activities in order to concentrate on this.                             | 1 | 2 | 3 | 4 |
| 56. I think hard about what steps to take.  | 1 | 2 | 3 | 4 |
| 57. I act as though it hasn't even happened.  | 1 | 2 | 3 | 4 |
| 58. I do what has to be done, one step at a time.   | 1 | 2 | 3 | 4 |
| 59. I learn something from the experience.  | 1 | 2 | 3 | 4 |
| 60. I pray more than usual.   | 1 | 2 | 3 | 4 |